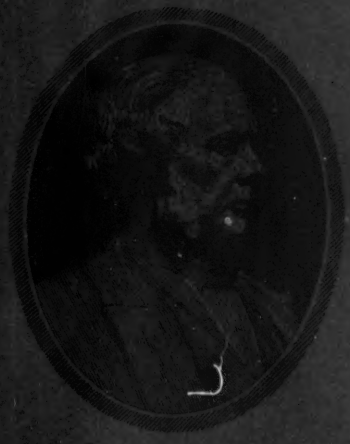
JUN 11 1947
MEDICAL LIBRARY

VOL. XXXIV QUARTERLY No. 135

The British Journal of Surgery



LISTER

JANUARY, 1947

BRISTOL: JOHN WRIGHT & SONS LTD.

LONDON: SIMPKIN MARSHALL (1941) LTD., 12, OLD BAILEY, E.C. 4

WHOLESALE DISTRIBUTORS: WM. DAWSON & SONS LTD.
43 WEYMOUTH STREET, LONDON, W.1.

UNITED STATES OF AMERICA: THE WILLIAMS & WILKINS COMPANY, BALTIMORE. *Sole Agents*

Entered as second class matter at the Post Office at BALTIMORE under the Act of March 3, 1879. (Sec. 225 P. L. & R.)

A & H Operation Table

Model E, on platform base, in lateral position, with lateral support, cushion, pelvic support, buttock support, and chest cushion.

Five models of the A & H Operation Table are made suitable for all the requirements of modern surgery.

LATEST IMPROVEMENTS include

Trendelenburg position increased to 75° tilt in model AE and 50° in model E; foot-operated Floor Brake, and rubber-tyred wheels fitted with self-oiling bearings. All models are easy to operate.

Descriptive booklet on application.

Model AE in extreme Trendelenburg position, tilt 75°. The shoulder rests are adjustable in both directions.

Over 1400 of these tables are in use at home and abroad.

ALLEN & HANBURYS LTD. LONDON, E.2

Makers of Quality Instruments

SHOWROOMS: 48, WIGMORE STREET, LONDON, W.1

THE
BRITISH JOURNAL OF SURGERY

VOL. XXXIV JANUARY, 1947 No. 135

DYSPLASIA EPIPHYSIALIS MULTIPLEX*

BY SIR THOMAS FAIRBANK

CASES in which several epiphyses have shown irregular ossification have been published from time to time under a variety of titles. Some are undoubtedly examples of one or other of the recognized conditions in which changes in some of the epiphyses constitute a minor feature: these will be referred to later. Others have been described as cases of multiple osteochondritis, with little or nothing, in my opinion, to support this diagnosis. With pseudo-coxalgia, if this be taken as a typical example of osteochondritis, these cases show nothing in common except irregular ossification and flattening of the head of the femur as seen in a radiograph. The progressive changes characteristic of pseudo-coxalgia —increased density followed by irregular decalcification, progressing to fragmentation, and succeeded eventually by recalcification of the ossific nucleus— have not been recorded in the cases under discussion.

It is the purpose of this paper to describe the features of a condition which I believe should be regarded as a clinical entity. I suggest it is essentially a developmental error resulting from some unknown cause. It is characterized by dwarfism, stubby digits, and mottling or irregularity in density and outline of several of the developing epiphyses, and it lacks the other abnormalities of the skeleton necessary for its allocation to one of the well-recognized groups. Whenever one of these groups is studied, atypical cases are invariably met with and difficulty is experienced in deciding whether to include an individual case in the group or relegate it to the scrap-heap of unclassified cases. This is true of the condition under discussion.

The following description is founded on the study of 20 cases, 9 of which have already been published, but only 2 of them under the title of this paper. The radiographs of a further unquestionable case were shown to me recently by Mr. N. W. Roberts. It is not claimed that the search of the literature has been exhaustive or that my decision to exclude a case has been invariably correct. Doubtful cases have been excluded.

The first case included was published by White in 1924 under the title of 'Chondro-dystrophia Fœtalis (Achondroplasia)'. The radiographic picture of achondroplasia is now so well known that this diagnosis can be questioned without hesitation. In 1930 Buxton showed at the Royal Society of Medicine a 'dwarf with stippled epiphyses'. In 1934 Jansen published a case which he labelled 'epiphysial dysostosis'. All three of these cases I believe to be examples of the condition described below. In 1937 Gardiner-Hill, in his Oliver-Sharpey Lectures, referred to two sisters as suffering from 'hereditary deforming dyschondroplasia'. I ventured to disagree with him over this diagnosis and Dr. Gardiner-Hill was good enough to furnish me with further details of these cases as well as of a boy he also referred to in the Lectures, but under a different title. All three of these cases, I believe should be placed in the group under discussion. Barrington-Ward, in 1912, published details of a brother and sister who had been under my care, and, although not included in this series, they are possible examples of this condition. In 1928 Grudzinski published two cases of atypical achondroplasia, or osteochondropathia multiplex, one of which at least appears to belong to the group under discussion. In 1943 Resnick published notes of a mother and her twin boys as cases of epiphysial dysplasia punctata: in my opinion, this diagnosis is unjustifiable, but I think they might well be placed in the multiplex group: they are not, however, included in my series. In 1935, in discussing the classification of generalized affections of the skeleton, I ventured to suggest 'epiphysial dysplasia' for the title of a group and briefly reported a case. To this title 'generalisata' was added, but this was altered later to 'multiplex', as being more accurate. In 1938 cases under this title were shown before the Royal Society of Medicine by Wiles and Yarrow respectively. There was some doubt at the time about the classification of the latter's case, but I think it should be included in the group.

The features of the condition found in the cases in this series are as follows: It is, of course, an affection of children and young people: the ages in this series varied from 6 to over 40 years. In one of Gardiner-Hill's cases the 'dyschondroplasia' was first noticed at the age of 18 months. One of my cases, first seen at 14 years, has been followed for over 20 years.

It affects both sexes (12 males, 8 females), and, as a rule, it is not inherited or familial. However, one case, a boy, appeared to have inherited it from

*A much abridged version of this article, then founded on 15 cases, was communicated to the Orthopædic Section of the Royal Society of Medicine in 1945. I am indebted to the Honorary Editors for permission to publish this enlarged and amended article elsewhere.

his mother, while two sisters are included in my series.

Mr. J. A. Cholmeley has furnished me with details of 2 cases, who are brothers and apparently the only ones affected in a family of ten. In another

FIG. 297.—Multiple epiphysial dysplasia in a boy (D. A.) aged 9 years. Hips showing irregular mottling of femoral heads. (Under Dr. K. M. Robertson.)

case many members of the family were unduly short, but it was impossible to examine them.

Intelligence is usually normal: in 2 cases, however, it was regarded as subnormal. Sexual infantilism was seen in only 1 case (Buxton's), the others being apparently normal in this respect. In 8 of the cases difficulty in walking was complained of, while in at least 5 others pain and stiffness of the knees or hips was the trouble which took them to hospital. In the older cases, as one would expect, arthritic symptoms are troublesome at times. Some degree of dwarfism, of the short limb type, is the rule, it being definitely recorded in 16 of the cases. The facies typical of cretinism is absent. Some enlargement of the epiphyses is occasionally seen. The hands are striking, the fingers and thumbs being short, thick, and stubby with blunt ends, the relative length of the digits being normal. Apart from the dwarfism and the shape of the hands, there is no characteristic deformity, but the following were noted: Flexion deformity of the knees was found in 3 cases, with subluxation present in 1 (*see below*). In 1 case the elbows were flexed and laxity of the wrists and knees was present. In another a curious deformity of the elbows was seen, the radial heads being subluxated. In yet another there was limitation of abduction of the shoulders and of extension of the elbows, while the knees were flexed and the tibial heads subluxated backwards. Genu valgum was present in 4, dislocation of the patella in 1, and bow-legs in another. In Jansen's case both hips were flexed, the knees flexed and valgoid, and the feet in the varus position. The spine is usually normal in length and ossification. The one exception to this rule was Jansen's case, which showed marked platyspondyly, with flattening and gross irregularity in outline of the vertebral bodies. Kyphosis was present in one and lordosis in another. Nothing special has been noted in the teeth: blood examinations have revealed nothing abnormal.

RADIOLOGICAL FINDINGS

The essential abnormalities are seen in the epiphyses. The centres of ossification for the epiphyses may be late in appearing and backward in development, and fusion with the shafts may be delayed, but the principle change is irregularity in ossification. They are irregular both in density and shape, being mottled in appearance and perhaps mulbery-like in outline. Separate subsidiary centres around the main centre are common, and this results in some peripheral stippling, but this is never carried

FIG. 298.—Multiple epiphysial dysplasia n a girl (P. R.) aged 7 years. Knees showing irregular ossification, with some stippling of femoral and tibial epiphyses, of moderate severity. Left patella is dislocated. (Under Mr. H. L-C. Wood.)

FIG. 299.—Ankles of same case as *Fig.* 298, showing irregular ossification and wedge shape of lower tibial epiphysis, and some corresponding alteration in shape of astragalus.

to the point seen in the punctate type of epiphysial dysplasia. There is a definite tendency towards improvement. As a rule, the epiphyses eventually become normal in density, but the outline, though usually smooth, remains permanently abnormal. Any or all of the epiphyses may be affected, but the most common to show typical changes and permanent deformity are the hips (*Figs.* 297, 302), shoulders, ankles, and less frequently the knees. The heads of the femora (and of the humeri) remain permanently shallow and less convex than the normal. In one exceptional case, a girl of 8 years, the femoral heads had already assumed a smooth flattened shape, without irregular density. The same was true of the humeral heads (*Figs.* 310, 311). In one typical case the femoral heads at the age of 27 still showed irregularity in outline and, on one side, partial fragmentation (*Fig.* 315), a condition that might well

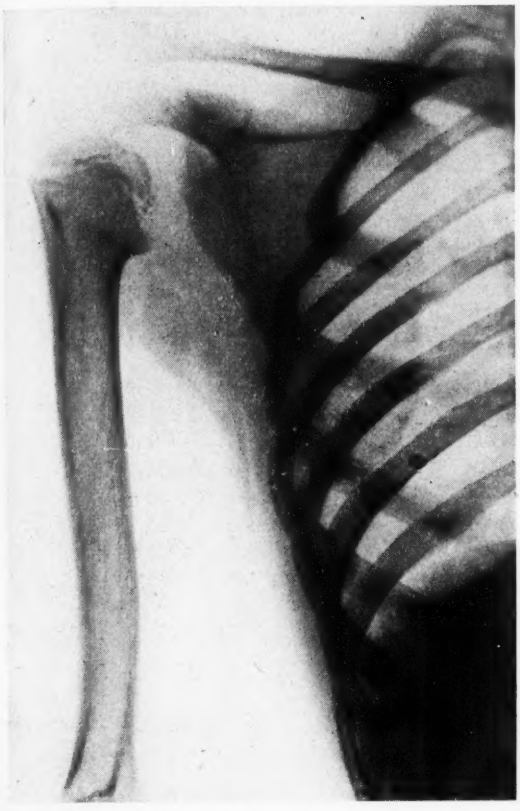


FIG. 300.—Multiple epiphysial dysplasia in a girl (J. F.) aged 9 years. Shoulders showing striking irregularity in ossification of upper epiphysis of humerus. Considerable stippling is present. Shaft of humerus is abnormally stout. (Under Mr. J. A. Cholmeley.)

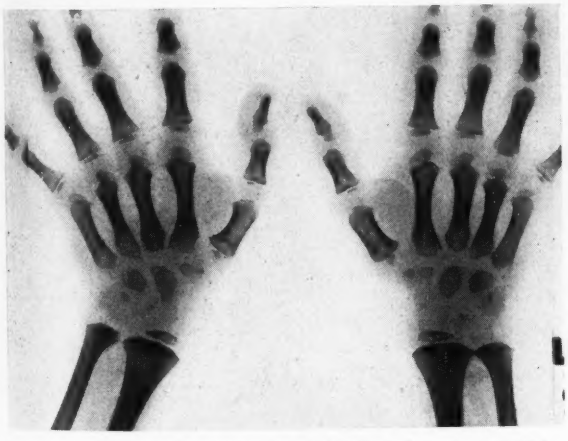


FIG. 301.—Multiple epiphysial dysplasia in a boy (J. H.) aged 7½ years. Wrists and hands showing backwardness of ossification of epiphyses of radius and ulna, and to some extent of the carpal bones, which are of irregular shape. Metacarpals and phalanges are shorter in comparison with their thickness than the normal.

FIG. 302.—Multiple epiphysial dysplasia in a boy (G. H.) aged 12 years. Hips showing retardation and irregularity of ossification of femoral heads and great trochanters. (Under Dr. H. Gardiner-Hill.)

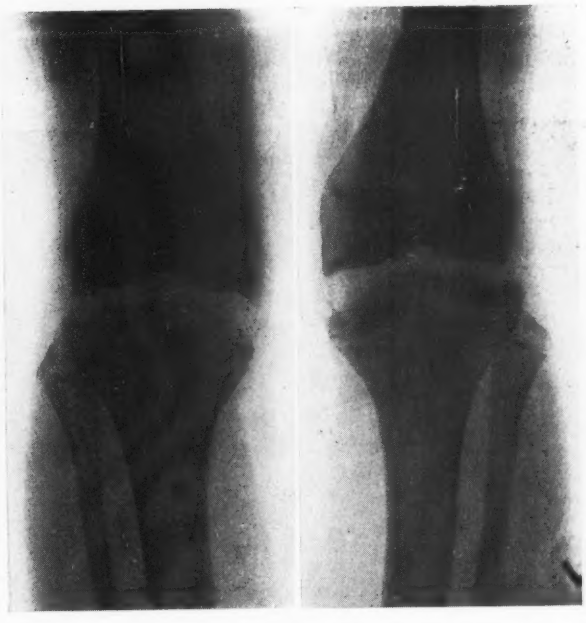


FIG. 303.—Knees of same case as *Fig.* 302, showing curious angular shape of femoral condyles and gross irregularity of ossification of inner tuberosities of tibiæ.

be mistaken for pseudo-coxalgia, which was, in fact, the diagnosis made when he was first seen at the age of 14. It is not surprising that in some of these cases this mistake should be made. The femoral neck may or may not be thickened. Some irregularity in shape of the acetabula was present in only 3 cases.

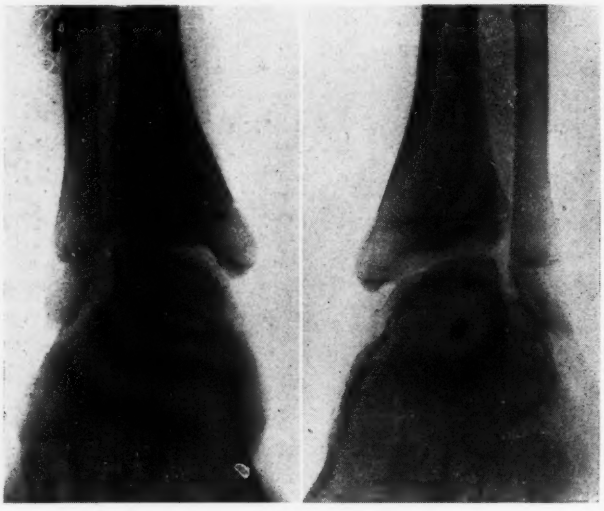


FIG. 304.—Ankles of same case as *Fig.* 302, showing striking deformity of lower tibial epiphyses and corresponding alteration in shape of astragali.

The great trochanter may be affected. The femoral condyles may show little more in the way of abnormal ossification than is seen from time to time in perfectly normal children (*Fig.* 298), the irregularity

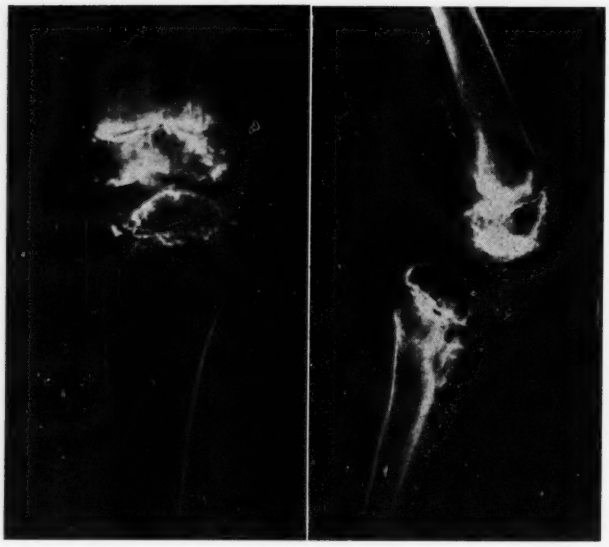


FIG. 306.—Multiple epiphysial dysplasia in a girl (J. S.) aged 13 years. Anteroposterior and lateral views of knee showing exceptionally marked irregularity in ossification of femoral condyle and tibial head. Some posterior subluxation of tibia on femur. (Under Mr. J. P. Campbell.)

in outline with separate points of ossification being particularly noticeable at the back of the condyles. In others the changes were much more marked (*Fig.* 306). In older children when ossification of the condyles is well advanced striking alteration in shape is seen in some cases (*Fig.* 303). The condyles in these cases are flattened and inclined to be

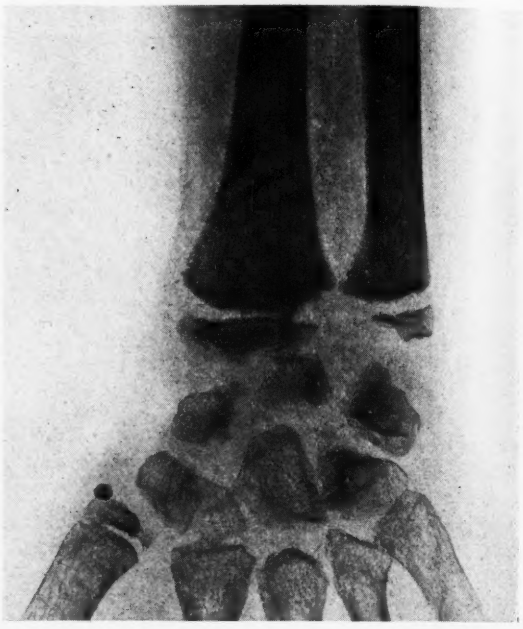


FIG. 305.—Wrist of same case as *Fig.* 302, showing gross irregularity in shape and density of the lower epiphyses of the radius and ulna and abnormal shape of the carpal bones.

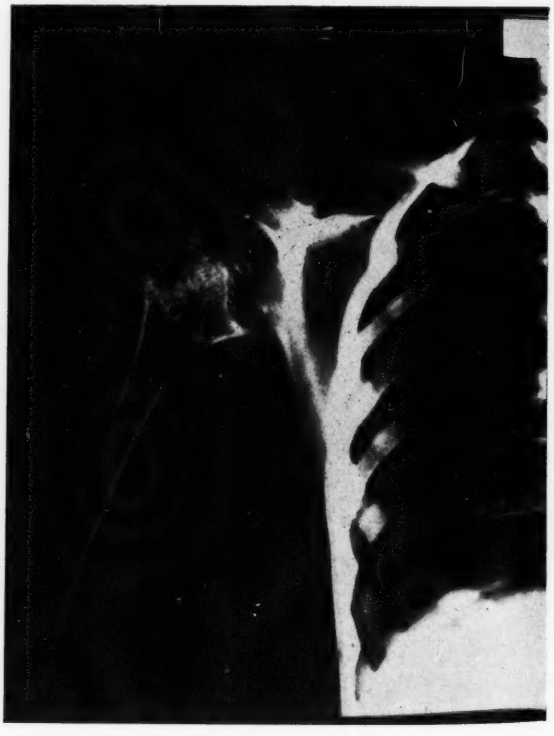


FIG. 307.—Shoulder of same case as *Fig.* 306, showing gross irregularity in ossification of upper humeral epiphysis and abnormalities in the shape and density of the metaphysis.

rectangular. The patellæ may show decided mottling. In one case the fibula was elongated, the head being placed unduly high.

The ankles show changes of diagnostic value. A certain amount of obliquity of the joint is met with not infrequently in apparently normal individuals—it has been seen also in cretins—but in the condition under discussion the obliquity is marked. The lower tibial epiphyses diminish in depth from within outwards to an exceptional degree. The joint line is therefore oblique, and the trochlear portion of the astragalus is misshapen to conform to this. This was a striking feature in at least ten of the cases (*Figs.* 299, 304, 312). The astragalus may or may not be tilted outwards. In three of the cases, in addition to that mentioned above, the fibula was too long, but in these it was the lower end of the bone that was displaced, the external malleolus lying abnormally low. There was no corresponding inequality in length of the radius and ulna in this series with one exception: the radius is relatively long in Wiles's case.

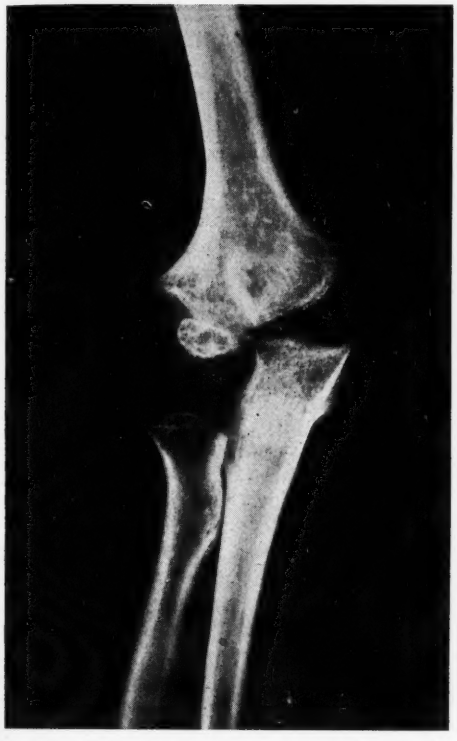


FIG. 308.—Elbow of same case as *Fig.* 306, showing delayed and abnormal ossification of epiphyses. Note the curious ossification of outer condyle, a dense nucleus for the capitellum being surrounded by less dense bone.

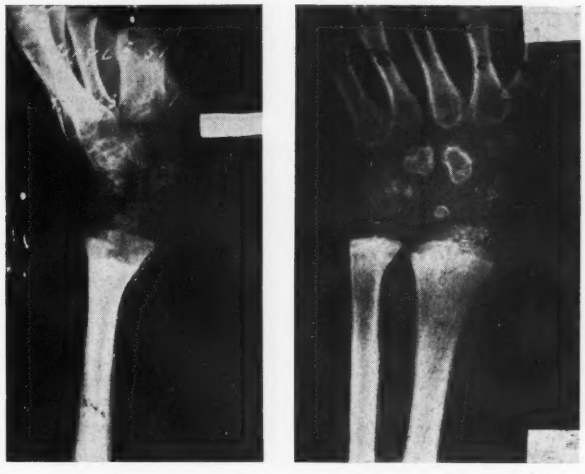


FIG. 309.—Wrist of same case as *Fig.* 306, showing delay and irregularity in ossification of radial and ulnar epiphyses and of the carpal bones. The abnormal appearances are particularly striking in this case.

The upper epiphysis of the humerus usually shows obvious irregularity in ossification with fragmentation (*Figs.* 300, 307) and sometimes the head appears to overlap the neck on the inner side to a striking degree (3 cases in this series). When ossification is advanced or complete the head remains flattened and less convex than usual (*Fig.* 314). Occasionally the head may show even greater permanent distortion (*Fig.* 317). The capitellum and distal radial epiphysis show definite changes in some cases (*Figs.* 301, 305, 309), but in the elbows and wrists no permanent deformity of importance has been noted with one exception, already referred to, in which there was subluxation outwards of the radial heads. In 3 cases the capitellum in each elbow shows in films a poorly calcified ossific centre,

FIG. 310.—Multiple epiphysial dysplasia in a girl (J. P.) aged 8 years. Shoulders showing marked flattening of humeral head.

FIG. 311.—Hips of same case as *Fig.* 310, showing shallow femoral heads, but without irregularity in density or fragmentation though child is only 8.

irregular in outline and with a sharply defined nucleus of much greater density (*Fig.* 308). The metaphyses show no consistent or characteristic change. In a few cases they were trumpeted, and the epiphysial lines irregular. In one some cystic-like change in the metaphyses was noted. The occasional overlap of the metaphysis by the humeral head on the inner side has already been referred to. In 1 case the epiphysis for the tibial head lay in an oblique anteroposterior plane and the tibia was subluxated backwards on the femur (*Fig.* 306).

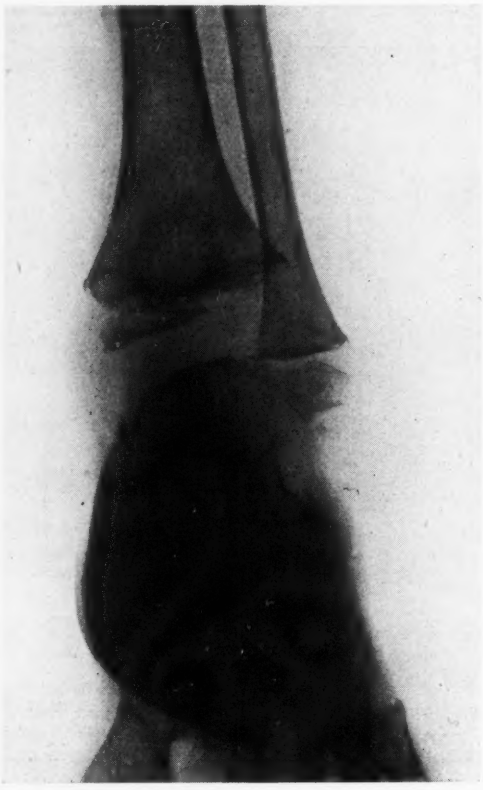


FIG. 312.—Multiple epiphysial dysplasia in a boy (D. B.) aged 6 years, showing typical abnormal ossification of lower tibial epiphysis, with some distortion of astragalus. There is some trumpeting of the metaphyses of tibia and fibula. (Under Mr. R. Broomhead.)

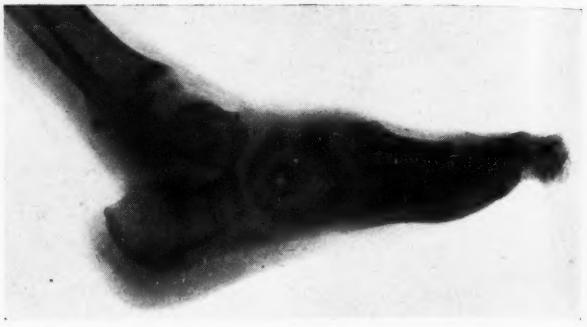


FIG. 313.—Foot and ankle of same case as *Fig.* 311, showing irregular shape of tarsal bones.

The shafts of the long bones are often shorter than normal, but not thickened as a rule (*Fig.* 300). Only in 1 case were they unduly curved. The carpal and tarsal bones ossify late, and may be very irregular in outline (*Figs.* 301, 305, 309, 313). The metacarpals, metatarsals, and phalanges are stunted (*Figs.* 301, 316). The epiphyses of some of these bones may show irregular ossification. In a few the metacarpals are rather suggestive of the Morquio-Brailsford type of osteochondro-dystrophy. The vertebral bodies show no peculiarities, the spine being normal, as a rule: only in 1 case already referred to were the vertebral bodies shallow and spread. The skull and teeth show no abnormalities.

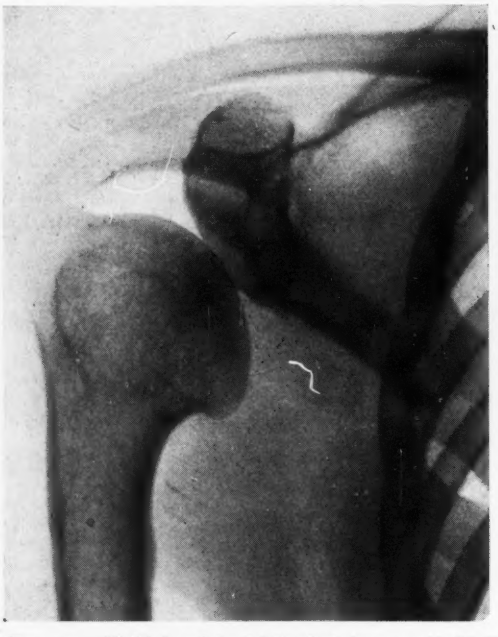


FIG. 314.—Multiple epiphysial dysplasia in a man (A. M. R.) aged 26, after being under observation for 12 years. Note flattening of humeral head and abnormal shape of glenoid. The other shoulder showed similar abnormalities.

FIG. 315.—Hips of same case as *Fig.* 314. Note irregular shape of both femoral heads, with partial fragmentation still present in the left. Arthritic symptoms in left hip were complained of at times.

The pathology is obscure. In Jansen's case a portion of the metaphysis, unfortunately not the epiphysis, at the lower end of a femur was examined microscopically. If I may venture an opinion on the published microphotograph I would say it shows mucoid degeneration.

FIG. 316.—Hand of same case as *Fig.* 314, showing typical blunt broad digits.

signs of this are absent, and there is no improvement in the appearance of the epiphyses in response to appropriate treatment. Two of the cases in our series were treated vigorously with thyroid, one for as long as two years, without obvious benefit. In cretins also the fingers are thick and stubby, but they fail to show their usual differences in length. Delay in fusion at the epiphysial line in cretins is associated with sclerosis of the terminal layer of the metaphysis. It is of interest to note that flattening of the humeral heads, strikingly similar to that seen in epiphysial dysplasia, has been seen in a cretin of 39 (Berard and Novel, 1930).

In dysplasia epiphysialis punctata, as it is now called, or stippled epiphyses, the *whole* of an epiphysis seems to be ossifying from a large number of discrete centres. The shafts of the long bones are short and thick, and the ends may be enlarged and splayed. The tarsal bones may be completely stippled, showing nothing but a collection of dots. The abnormalities generally are much more gross than in most cases of the multiplex group, and no difficulty should be experienced in differentiating the two. Congenital cataract occurs in about half the cases.

In chondro-osteo-dystrophy of the Morquio-Brailsford type, but not in gargoylism as a rule, the femoral heads show striking epiphysial changes, but in this condition the acetabula are markedly enlarged and irregular. Other epiphyses and the carpal and tarsal bones frequently show irregularity in ossification, but notable features are the abrupt kyphotic deformity in the dorsilumbar region, resulting from the diminution in size of one vertebral body, which is displaced backwards, and the shape of the vertebral bodies; in the latter the central prolongation anteriorly is quite distinctive and diagnostic.

In dyschondroplasia changes in the epiphyses, when seen, are of no importance, being completely

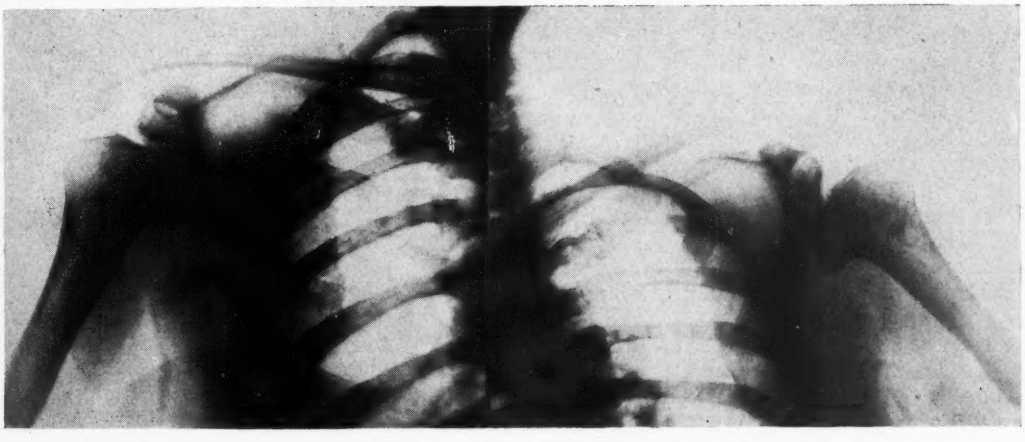


FIG. 317.—Shoulders of a middle-aged woman, showing unusually marked changes in shape of the humeral heads and glenoid cavities. The changes seen in other joints strongly suggest this case is one of multiple epiphysial dysplasia. (*By courtesy of Dr. H. Austin Williams.*)

DIFFERENTIAL DIAGNOSIS

There are at least seven other conditions, besides osteochondritis, in which the epiphyses may show irregular ossification. Obviously cretinism is the first general condition to be excluded. The classical

overshadowed by the gross abnormality of the metaphyses; irregularity in density of an epiphysis is never seen except when the adjacent portion of the metaphysis contains obvious masses of cartilage. Dyschondroplasia is usually, but not invariably, unilateral in distribution. The affected portions of

the bones, i.e., the chondromatous areas, become stippled with dense spots in the later or recovering stages of the condition. In that skeletal curiosity called osteopoikilosis, the epiphyses are of normal shape, and, whether fused to the shaft or not, show large numbers of the characteristic discrete dense spots, though these are by no means confined to the epiphyses. In osteopetrosis or marble bones, some of the epiphyses may show irregular density or stippling, but the changes in the shafts dominate the picture.

Lastly, mottled epiphyses have been described in association with pituitary gigantism (Traub, 1939). Some mottling, particularly of the femoral heads, has been met with very occasionally in other endocrine errors and in cases which at present it is impossible to classify.

Finally, I would suggest that if there is reason to suspect pseudo-coxalgia in a patient below the average in height, it is well worth while having films taken of the shoulders and ankles to exclude the developmental error which is the subject of this paper.

I am greatly indebted to the following friends who have supplied me with details of cases under their care: Mr. R. Broomhead, Dr. G. T. Calthrop, Mr. J. P. Campbell, Mr. J. A. Cholmeley, Dr. E. A. Cockayne, Dr. H. Gardiner-Hill, Mr. A. D. Le Vay, Dr. K. M. Robertson, Mr. V. C. Snell, Dr. H. Austin Williams, and Mr. H. L-C. Wood.

REFERENCES

BARRINGTON-WARD, L. E. (1912), *Lancet*, **1**, 157.
BERARD AND NOVEL (1930), *J. Radiol. Electrol.*, **14**, 329.
BUXTON, ST.J. D. (1930), *Proc. R. Soc. Med.* (Orth. Sect. 53), **23**, 1329.
FAIRBANK, H. A. T. (1935), *Ibid.* (Clin. Sect. 47), **28**, 1611.
GARDINER-HILL, H. (1937), *Brit. med. J.*, **1**, 1241.
GRUDZINSKI, Z. (1928), *Fortschr. Röntgenstr.*, **38**, 873.
JANSEN, M. (1934), *Z. orthop. Chir.*, **61**, 3 H., 253.
RESNICK, B. E. (1943), *J. Bone Jt Surg.*, **25**, 461.
TRAUB, E. (1939), *Arch. Dis. Childh.*, **14**, no. 79, 203.
WHITE, R. (1924), *Brit. J. Surg.*, **12**, July, 81.
WILES, P. (1938), *Proc. R. Soc. Med.* (Orth. Sect. 7), **32**, 279.
YARROW, D. E. (1938), *Ibid.* (Child. Sect. 11), **32**, 315.

SARCOMA IN ABNORMAL BONES

BY HARRY PLATT
PROFESSOR OF ORTHOPÆDIC SURGERY, UNIVERSITY OF MANCHESTER

IN a personal series of 180 fully documented primary malignant tumours of bone (period 1920–45), of 161 tumours belonging to the sarcoma group, 18 are sarcomas occurring in abnormal bones. This sub-group, which comprises: (1) 10 sarcomas occurring in Paget's disease, (2) 2 sarcomas arising in fibrocystic disease, and (3) 6 tumours representing the malignant transformation of a previously benign osteochondroma, presents a number of instructive features which illustrate the wide variations in the behaviour pattern of bone sarcoma.

1. PAGET SARCOMA

The incidence of sarcoma in osteodystrophia deformans has been estimated by various observers as between 5 and 10 per cent. Paget himself was aware of the liability of the victims of the disease which later was to bear his name to develop malignant tumours, but of the 5 cases he reported, in only 2 did the tumour arise definitely in connexion with bone (1889).

Of my own cases, 8 were males and 2 females. The malignant tumours appeared in 4 patients between the ages of 50 and 60, and in 6 patients over 60. Of the bones affected: 4 tumours involved the femur (lower end, 3; upper end, 1); 4 the humerus (upper end, 2—in the same patient; and shaft, 2); and 3 were pelvic tumours (ilium, 2; pubis, 1). In the majority of the cases there was a fairly long prodromal stage in which Paget's disease had manifested either in one bone or throughout a considerable part of the skeleton before the appearance of the tumour. The histological type of tumour in all cases was a spindle or polymorphic sarcoma.

Two additional cases have recently come under my care (1946)—both females—one a sarcoma of the upper end of the humerus arising in a somewhat atypical form of Paget's disease with predominantly

FIG. 318.—*Case* 1. M., aged 63. Paget's disease. Large sarcoma of lower end of femur.

osteoporotic changes in the affected bones (*Figs.* 332, 333); the second case, a woman of over 70 with generalized Paget's disease of long standing, with multiple tumours in the frontal and parietal areas of the skull.

In my experience these Paget sarcomas have proved to be highly lethal. Seven patients died within the year following the discovery of the tumour

FIG. 319.—Same case. Gross changes in right half of pelvis and lumbar spine

and its treatment. But 1 patient is still alive (1946) two years after disarticulation at the shoulder for a sarcoma of the upper end of the humerus (*Case* 5).

Seven patients were treated by radical operations: 3 by disarticulation at the shoulder-joint; 2 by amputation through the thigh; 1 by disarticulation at the hip; and 1 by a *hindquarter amputation*—the last being one of a series of 6 patients in whom I have carried out this formidable procedure during the past eight years for the treatment of malignant tumours of the bony pelvis. In the inaccessible tumours irradiation was used with no appreciable effect on the tumour or benefit to the patient in general.

ILLUSTRATIVE CASES

Case 1.—**Male, aged 63. Sarcoma of lower end of femur (L.).**

HISTORY.—Pain in left knee with increasing stiffness—five years' duration. Three weeks before entering hospital knee became much more swollen after a fall. Loss of weight during last few months.

ON EXAMINATION (Aug. 16, 1938).—Looks thin and worn. Left knee almost fixed in 90° flexion with large mass incorporated with lower end of femur extending round to popliteal region (*Fig.* 318). Spine: rounded dorsal kyphosis.

X-ray Examination.—Gross changes of Paget's disease in pelvis (R. half only); lumbar spine. Skull: normal. Left femur (lower half): Paget's changes with destruction of lower third of shaft and soft-tissue shadow filling popliteal fossa. (*Figs.* 319, 320.)

TREATMENT.—Amputation of upper third of thigh on Aug. 26.

AFTER-TREATMENT.—Coley's toxins for three weeks. X-ray therapy.

HISTOLOGY.—Spindle-cell sarcoma (*Fig.* 321).

RESULT.—Death 3½ months after amputation.

Comment.—Long prodromal period. Irregular distribution of Paget's disease in skeleton. Short survival after amputation.

Case 2.—**Female, aged 60. Sarcoma of lower end of femur (L.).**

HISTORY.—Seven years ago sustained subtrochanteric fracture of left femur. Three months ago left knee became swollen.

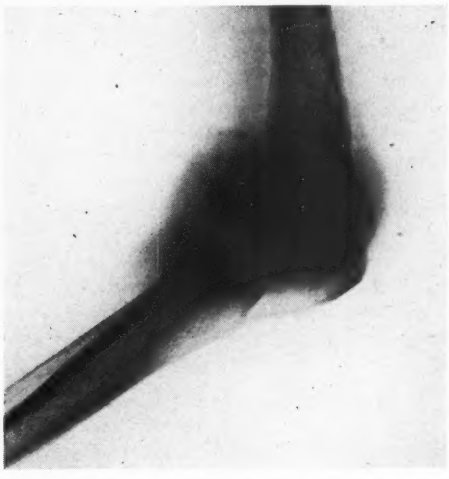


FIG. 320.—Same case. Showing destructive changes and soft-tissue shadow.

ON EXAMINATION (Aug. 31, 1937).—L. knee: enormous swelling and enlargement of lower end of femur with dilated veins in overlying skin (*Fig.* 322).

X-ray Examination.—Changes of Paget's disease in skull, left femur, and pelvis; localized destruction of

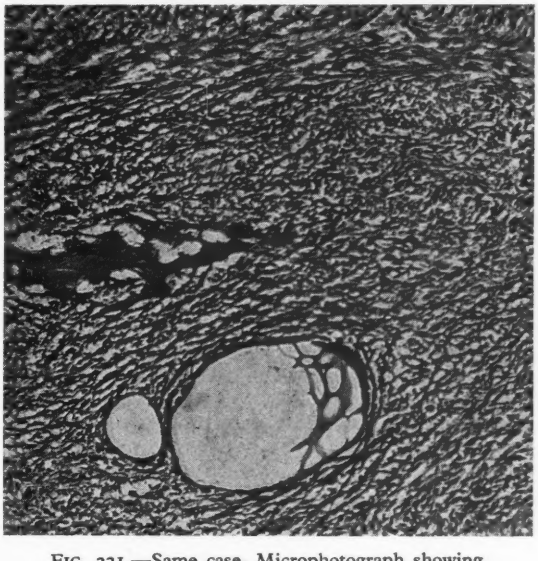


FIG. 321.—Same case. Microphotograph showing spindle-cell sarcoma. (× 40.)

lower end of femur and shadow of large extra-osseous tumour (*Figs.* 323, 324).

TREATMENT.—Disarticulation of left hip on Jan. 10, 1938.

HISTOLOGY.—Spindle-cell sarcoma.

RESULT.—Patient died six days later with signs of renal failure.

Comment.—Long-standing Paget's disease. Massive sarcoma involving lower end of left femur with

FIG. 322.—*Case* 2. F., aged 60. Paget's disease. Sarcoma lower end of femur. Large tumour. Note discoloration and dilated veins.

FIG. 323.—Same case. Shows Paget's disease of pelvis and left femur.

FIG. 324.—Same case. Shows massive destruction.

FIG. 325.—Same case. Amputation specimen showing two tumour masses: (1) lower end of femur, and (2) upper end of femur—representing spread along fascial planes.

spread upwards along fascial planes to the region of the great trochanter, where there was another large mass. Tumour *not* apparently of multicentric origin (*Fig.* 325).

FIG. 326.—*Case* 3. M., aged 53. Paget's disease. Sarcoma of pelvis. Shows Paget's changes in pelvis and femur, with destruction in pubic ramus and soft-tissue shadow.

Case 3.—**Male, aged 53. Sarcoma of pelvis (L.).**

HISTORY.—Pain for six weeks, gnawing in character, on lateral aspect of left knee, worse at night. Recently noted painful lump in left groin.

ON EXAMINATION (Oct. 4, 1939).—L. thigh: large irregular mass in Scarpa's triangle firmly attached to pubis and pubic ramus. Hip movements: slight limitation of flexion and abduction.

X-ray Examination.—(1) Paget's changes in pelvis, lower spine, and left femur—dense osteosclerosis. (2) Area of destruction in left pubic ramus, with soft-tissue shadow—pathological fracture through ramus (*Fig.* 326).

TREATMENT.—Hindquarter amputation, left, Oct. 13 (*Fig.* 327).

SUBSEQUENT COURSE.—Three months later mass observed in left erector spinæ.

X-ray Examination.—Pelvis: extensive destruction of sacrum. Spine: deposit in 3rd lumbar vertebra. Chest: secondary deposits.

RESULT.—Death three and a half months after hindquarter amputation.

Comment. — Pre-existing Paget's disease of long duration. Short survival following hindquarter amputation.

Case 4.—**Male, aged 61. Sarcoma of upper end of humerus (R. and L.).**

HISTORY.—

Sept. 16, 1938: Attended fracture clinic after fall on to *right* shoulder. Radiograph showed crack across surgical neck of humerus with appearances suggesting Paget's disease. Recovery of almost full range of movements in shoulder.

Dec. 4, 1939: Admitted to hospital with swelling upper end of right humerus.

ON EXAMINATION.—Swelling right shoulder and upper third arm; limitation of movement at shoulder-joint.

X-ray Examination.—Humerus (R.): Areas of dense bone with mottling; new bone, outer side of head and

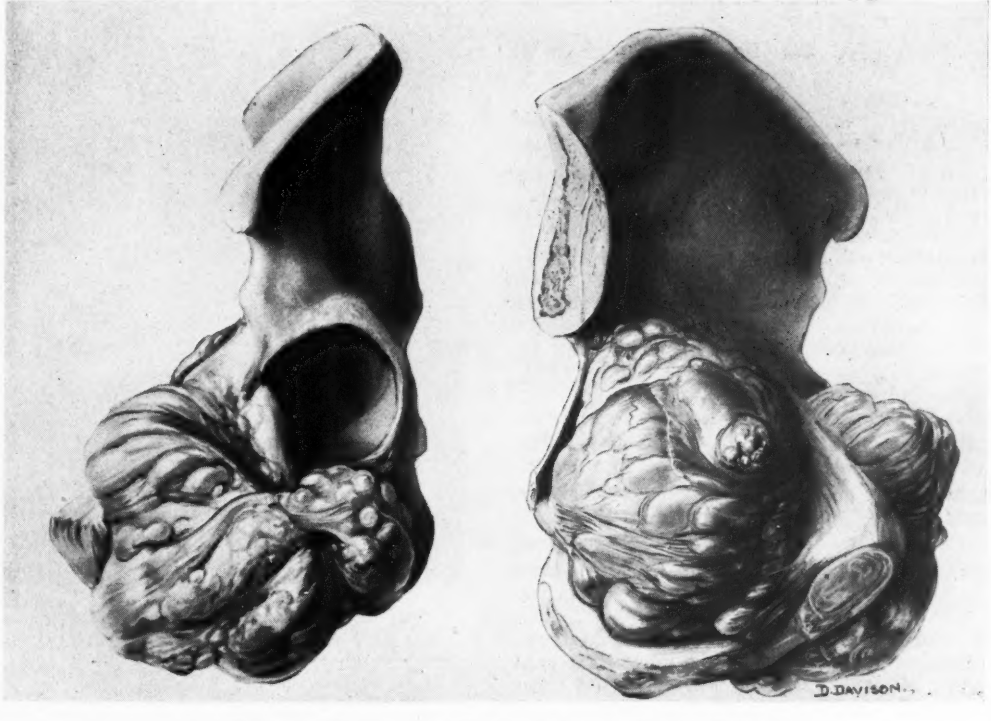


FIG. 327.—Same case. Hindquarter amputation specimen.

neck of humerus (*Fig.* 328). Lumbar spine and pelvis: no changes.

Biopsy.—Dec. 20, 1939.

TREATMENT.—Disarticulation of shoulder (R.).

HISTOLOGY.—Spindle-cell sarcoma.

FIG. 328.—*Case* 4. M., aged 61. Paget's disease. Sarcoma of upper end of humerus (R. and L.). Tumour of upper end of humerus (R.).

PROGRESS.—

Jan. 11, 1940: Local recurrence in amputation scar in *right* axilla. Put on deep X-ray therapy. Mass remained stationary in size.

Nov. 26.: Tumour mass now discovered in *left* shoulder region; mass in right axilla not increased in size. Radiography: massive destruction of upper end of *left* humerus and scapula (*Fig.* 329).

RESULT.—Death (Jan. 26, 1941).

Comment.—Paget's disease of some years' standing. *Two* separate tumours arising in the upper end of each humerus. Short survival following disarticulation of the upper limb for the sarcoma first discovered. An example of *multicentric* tumour formation.

Case 5.—**Male, aged 67. Sarcoma of shaft of humerus (R.).**

HISTORY.—

June 19, 1942: While digging in the garden, fell and right arm "gave way"—fracture humerus diagnosed—changes of Paget's disease not recognized at that time. Fracture united in three months.

May, 1944: Returned complaining of pain and swelling in right arm of three days' duration.

ON EXAMINATION.—R. arm: mass in upper third involving humerus.

X-ray Examination.—Paget's changes with localized destruction (*Fig.* 330).

TREATMENT.—Disarticulation at shoulder (June 19, 1944) (*Fig.* 331).

HISTOLOGY.—Polymorphic-cell sarcoma.

SUBSEQUENT COURSE.—Well; no signs of local recurrence or secondaries elsewhere two years later (June, 1946).

Comment.—Long prodromal stage before development of tumour. Survival two years following amputation.

Case 6.—**Female, aged 62. Sarcoma of humerus (L.).**

HISTORY AND SIGNS.—

1944: Extensive changes in upper third of humerus (L.) with restriction of movement at shoulder discovered after two years of pain. Radiographic changes compatible with Paget's disease, but porotic changes predominant.

Throughout 1945 severe pain which defied all forms of drug therapy.

December, 1945: Radiography showed destructive changes in head of humerus suggesting malignant change (*Fig.* 332).

FIG. 329.—Same case. Second tumour in the same patient, upper end of left humerus.

TREATMENT.—Irradiation with no relief followed by discovery of spontaneous fracture with gross destructive changes, but no palpable extra-osseous tumour.

FIG. 330.—*Case* 5. M., aged 67. Paget's disease. Sarcoma of shaft of humerus. Extensive Paget's disease in humerus with tumour in middle of shaft.

March 11, 1946: Disarticulation at shoulder (*Fig.* 333).

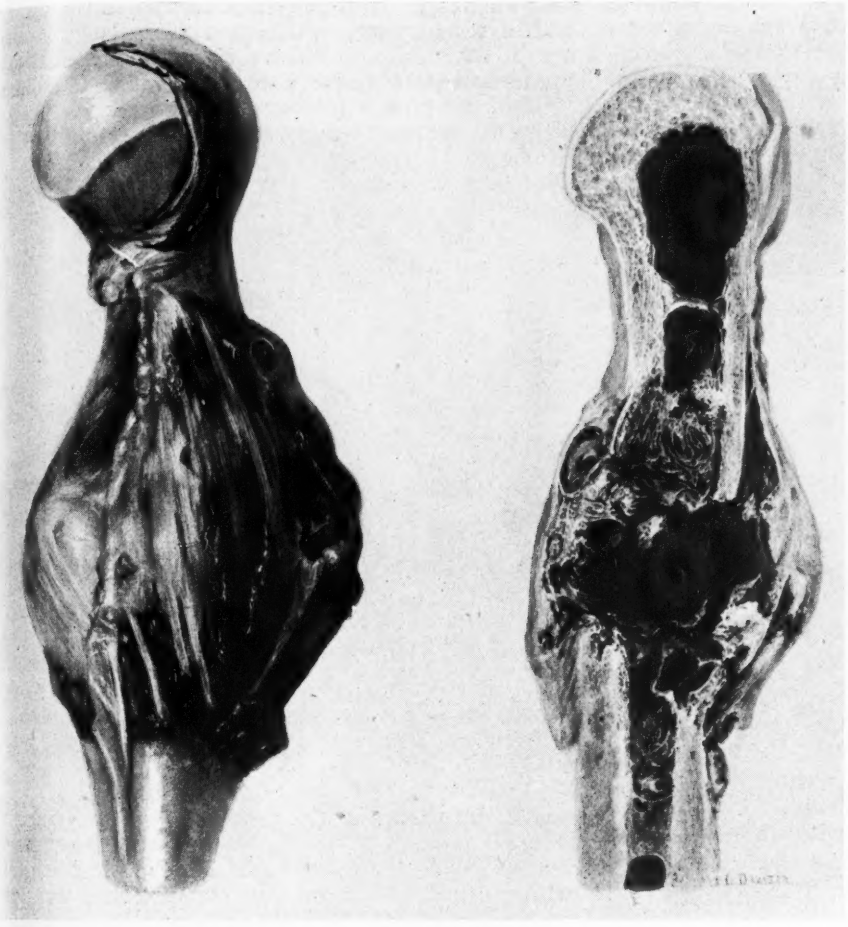


FIG. 331.—Same case as *Fig.* 330. Amputation specimen.

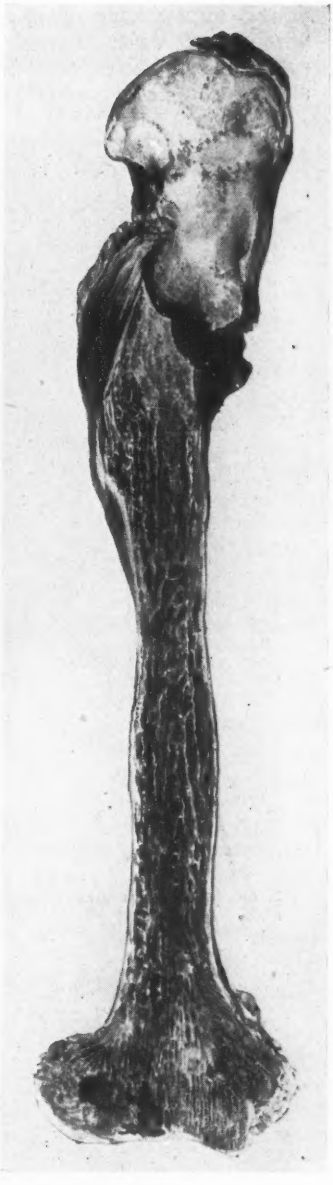


FIG. 333.—Same case as in *Fig.* 332. Amputation specimen. Note predominantly osteoporotic change.

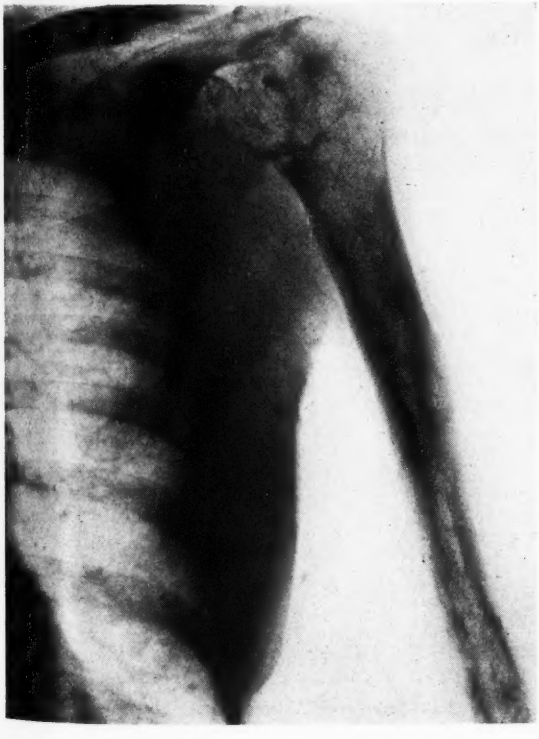


FIG. 332.—*Case* 6. F., aged 62. Paget's disease. Sarcoma of upper end of humerus (L.). Extensive Paget's changes in humerus, with expansile lesion occupying head and neck of the bone.

HISTOLOGY.—Spindle-cell sarcoma.

Comment.—Case presents two unusual and striking features: (1) A predominantly osteoporotic type of Paget's disease; (2) A true endosteal or central sarcoma with no evidence of extra-osseous tumour.

2. SARCOMA IN FIBROCYSTIC DISEASE

Sarcomatous transformation in a benign cyst or focal area of osteodystrophia fibrosa would appear to be a very rare phenomenon (Coley and Stewart, 1945). The 2 examples in the present series were sarcomas arising in dystrophic cysts after comparatively long intervals. The first case was the malignant transformation of a large cyst of the ilium in a

young man under observation for seven years in whom, after death, a large sarcoma was found completely filling the pelvis, but with no trace of secondary deposits elsewhere (*Figs.* 334–337). The second case was a man with a dystrophic cyst in the upper end of the humerus which, ten years later, at the age of 48, began to behave as a tumour and was found at biopsy to be a fibrosarcoma. The limb was disarticulated at the shoulder, and the patient is known to be alive at the present time, nine years after the operation. A subsequent histological study of material from the amputation specimen has confirmed the original diagnosis.

The experience of these two rare cases reinforces one's belief in the wisdom of obliterating dystrophic cysts in adults by appropriate operative procedures (curettage and the implantation of bone-grafts).

FIG. 334.—M., aged 18. Fibrocystic disease of pelvis. Shows large cyst in ilium after curettage and insertion of bone-graft. Histological examination of cyst contents showed changes of fibrocystic disease only.

FIG. 336.—Same case. Condition four years later. Almost complete destruction of bony pelvis on right side, with further displacement of femur. Extension of lesion into sacrum on right side. Biopsy at this stage revealed sarcomatous change.

FIG. 335. Same case. Condition eleven months later. Shows extension of cyst and displacement of head of femur

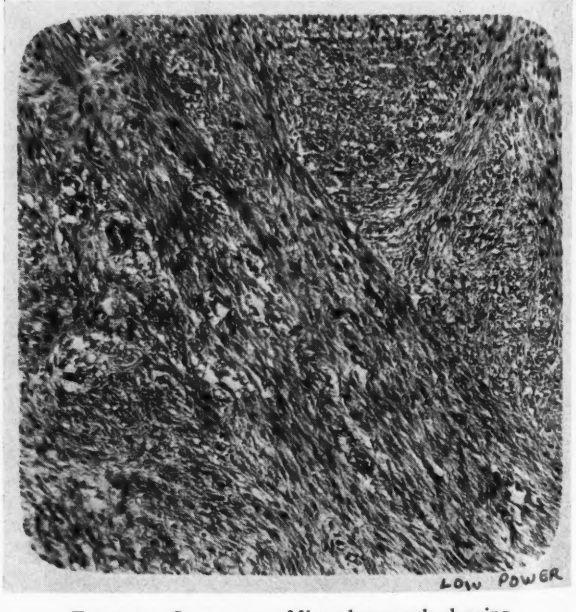


FIG. 337.—Same case. Microphotograph showing spindle-cell sarcoma.

3. MALIGNANT TRANSFORMATION IN BENIGN OSTEOCHONDROMA

The 6 examples of malignancy supervening in benign osteochondroma represent a type of tumour whose characteristics are fairly well known. Three of these tumours appear in my recorded series of 23 five-year survivals in various types of bone sarcoma (Platt, 1947). The recognition of the advent of malignancy in a chondroma or osteochondroma is not always an easy matter, and more especially in tumours with a long history which later grow to an enormous size, and which may be relatively inaccessible, as for example when involving the bony pelvis (*Fig.* 338). Paget pointed out long ago (1853) that rapid growth and large size are not in themselves criteria of malignancy, and the truth of this observation is borne out in the recent study of osteochondromata of the pelvic bones by Ghormley, Meyerding, Mussey, and Luckey (1946). Nevertheless, a presumed benign osteochondroma which begins to increase in size should be effectively removed before it reaches the dangerous stage of transition into malignancy.

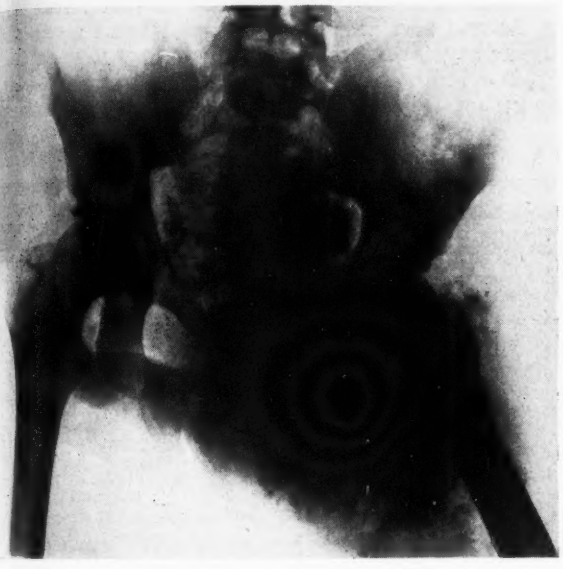


FIG. 338.—M., aged 24. Osteochondroma of pelvis. Enormous osteochondroma. No histological proof of malignant change.

SUMMARY

1. A study of 18 sarcomas arising in abnormal bones is presented.

2. The prognosis in the sarcomas of Paget's disease would appear to be grave. In 7 out of 10 cases in the series the patients died within a year from the discovery of the tumour and its treatment. One patient only, a man of 67 with a sarcoma of the humerus, has so far survived two years from the time of amputation.

3. The appearance of sarcoma in an area of fibrocystic disease is a rare phenomenon. In the 2 recorded cases the survival periods were two years and nine years respectively. In the former, an inoperable tumour of the pelvis of gigantic size, at the time of death no evidence of metastasis was revealed.

4. In the type of sarcoma resulting from the malignant transformation of an osteochondroma (6 cases in the series), examples of long survival periods are likely to be encountered. Although rapid growth and the attainment of enormous size are not in themselves conclusive evidence of the advent of malignant change, tumours which behave in this fashion should be eradicated without delay.

BIBLIOGRAPHY

COLEY, B. L., and STEWART, F. W. (1945), *Ann. Surg.*, **121**, No. 6, June.

GHORMLEY, R. K., MEYERDING, H. W., MUSSEY, R. D., and LUCKEY, C. A. (1946), *J. Bone Jt Surg.*, **28**, No. 1, Jan.

PAGET, Sir JAMES (1853), *Lectures on Surgical Pathology*. London.

— — (1889) "Remarks on Osteitis Deformans", *Illus. med. News.*, **2**, No. 181.

PLATT, HARRY (1947), "Survival in Bone Sarcoma" (in process of publication).

General :—

BIRD, CLARENCE E. (1927), "Sarcoma complicating Paget's Disease of the Bone", *Arch. Surg.*, **14**, No. 6, June.

DAVIE, T. B., and COOKE, W. E. (1937), "The Supervention of Osteogenic Sarcoma in Paget's Disease", *Brit. J. Surg.*, **25**, No. 98, Oct.

GESCHICKTER, C. F. (1931), "Fibrocartilaginous Tumors of the Bone", *Arch. Surg.*, **23**, No. 2, Aug.

— — and COPELAND, MURRAY M. (1929), "Osteitis Fibrosa and Giant-cell Tumour", *Ibid.*, **19**, No. 2, Aug.

NEEDLE BIOPSY IN THE CLINICAL DIAGNOSIS OF TUMOURS

By FRANK ELLIS

DIAGNOSIS BY NEEDLE BIOPSY

WITHOUT histological evidence the true nature of a neoplasm may be unknown and the critical attitude to published series of results of treatment, especially by radiotherapy, without this evidence is such that it must be considered essential. Clinical findings with radiological and laboratory evidence unsupported by histology may often be misleading and doubt of the diagnosis becomes especially marked when five-year cures by radiotherapy are used to assess the value of treatment.

Khanolkar and Sharpure (1944) show in a review that in various surgical clinics clinical diagnosis was wrong in from 10 to 57 per cent of cases as compared with the subsequent histological diagnosis. Biopsy from an ulcerated surface has no serious dangers, as shown by various authors. Marzloff (1923) showed that 36·8 per cent of 38 long-surviving cases of cancer of the cervix uteri had a diagnostic curettage several days before operation. Haagensen and Stout (1942) found 49 per cent five-year cures with biopsy as against 34 per cent without. Paterson and Nuttall (1939) reported similar findings in accessible carcinoma of the tongue and lip.

Needle biopsy causes less disturbance and therefore, on *a priori* grounds, less danger than other methods for non-ulcerated lesions. Coley et al. (1931) found no untoward effects in bone tumours, and Martin and Stewart (1936) report in similar manner on 3500 cases of aspiration biopsy, including many cases of visceral puncture and 60 cases of cervical lymph-node metastasis free of disease from 1 to 6 years following treatment by radiation.

Aspiration biopsy as an aid to rapid diagnosis of malignant neoplasms has been developed principally by Martin and Ellis (1930), since when its use and results have been reported by them (1934, 1940) and by many other workers (Ferguson, 1930; Coley, Sharp, and Ellis, 1931; Stewart, 1933; Binkley, 1939; Sayago, 1942; Iversen and Roholm, 1939; Ottolenghi, 1943).

Table I.—ASPIRATION BIOPSIES. CHRONOLOGICAL ANALYSIS

	Malignant Correct	Malignant Incorrect	Non-malignant Correct	Non-malignant Incorrect	No Result —Later Malignant	No Result —Fate Unknown	Diagnosis Unknown	No Result —Later Non-malignant	Total	Percentage of Useful Biopsies
1936	7 (50 per cent)	0	4 (29 per cent)	0	2	1	0	0	14	79
1937	21 (47 per cent)	2	3 (7 per cent)	0	10	8	0	1	45	54
1938	57 (65 per cent)	4	7 (8 per cent)	3	4	9	0	3	87	73
1939	59 (59 per cent)	0	9 (9 per cent)	0	8	13	7	4	100	68
1940	46 (52 per cent)	0	9 (10 per cent)	1	5	19	2	6	88	62
1941	70 (51 per cent)	2	15 (11 per cent)	4	20	17	3	6	137	62
1942	98 (50 per cent)	0	14 (7 per cent)	3	34	42	4	0	195	57
1943	10 (43 per cent)	0	4 (17 per cent)	0	3	5	1	0	23	60
	368 (54 per cent)	8 (1·2 per cent)	65 (9 per cent)	11 (1·6 per cent)	86 (13 per cent)	114 (15 per cent)	17 (2·5 per cent)	20 (3 per cent)	689	63

Table II.—ASPIRATION BIOPSIES. ANALYSIS OF RESULTS FOR CERTAIN SITES

	Malignant Correct	Malignant Incorrect	Non-malignant Correct	Non-malignant Incorrect	No Result —Later Malignant	No Result —Fate Unknown	Diagnosis Unknown	No Result —Later Non-malignant	Total	Percentage of Useful Biopsies
Glands	151	3	26	10	32	44	9	5	280	63
Bone	48	1	4	0	12	12	7	3	87	60
Breast	49	1	14	3	15	19	3	4	108	58
Intrathoracic	10	0	0	0	3	4	0	1	18	56
Abdominal	13	0	1	0	6	2	0	0	22	64
Prostate	5	0	0	0	0	5	0	0	10	50
Kidney	2	0	0	0	0	0	0	0	2	100
Totals	278	5	45	13	68	86	19	13	527	61·3

Indications for Needle Biopsy.—These are:—

1. When a tumour lies deep to normal skin or other tissues.

2. When the possibility exists of the tumour being infected.

3. When a major operation would otherwise be necessary and when such an operation would not allow of the successful extirpation of the tumour.

4. When usual methods of obtaining histological evidence are contra-indicated.

5. When excision of a lymph-node is contemplated in a suspected case of Hodgkin's disease, needle biopsy may exclude cancer and may establish a diagnosis of Hodgkin's disease.

The experience which I have had with aspiration biopsies has been sufficiently encouraging to justify its frequent use. Many of the unproductive biopsies were attempted on lesions of doubtful nature and even with a doubt in some cases of the existence of a pathological process. The final tables were prepared by comparing the result of the aspiration biopsy with the clinical notes based on an efficient follow-up system.

RESULTS

Aspiration Biopsy.—*Table I* shows the chronological distribution of the aspiration biopsies carried out by the author at the Sheffield Royal Infirmary and Royal Hospital, Sheffield, with an indication of the percentage distribution of the useful biopsies (i.e., malignant correct plus non-malignant correct). The figures for the years 1936 and 1943 do not cover the whole year in either case because the method was first adopted in 1936 and because I left Sheffield at the end of April, 1943.

It would appear that the greater the number of aspiration biopsies taken in any one year, the greater tends to be the 'wastage', since in 1941 and 1942 the percentages of useful results, corresponding to 137 and 195 biopsies respectively, were 62 per cent and 57 per cent. The reason for this may be the greater confidence developed in attempting biopsies on many kinds of unpromising lesions, although at all times the decision to carry out the examination is made because of the necessity for determining the histology of a lesion and the difficulties of varying kinds in making such determination by a more drastic surgical approach.

Table II shows an analysis of biopsies from several sites indicating the number of definite diagnoses and the percentages of useful diagnoses for these sites.

In the case of lymphatic glands the biopsies were mostly taken from the neck, as shown by *Table III*.

Table III.—Aspiration Biopsies
Regional Lymph-glands from which Biopsies Taken

Region	*No. Taken*
Axillary	16
Neck	176
Supraclavicular	26
Submaxillary	16
Groin	28
Iliac	3
Submental	1
Unknown or other regions	14
Total	280

In the case of lesions of bone the biopsies were mostly taken from the femur and the mandible, as shown in *Table IV*.

The distribution of the correct malignant diagnoses, according to the histology and the site from which the biopsy was taken is listed in *Table V*. No attempt is made in this table to distinguish between primary and metastatic lesions.

Table IV.—Aspiration Biopsies
Bones from which Biopsies Taken

Name of Bone	*No. Taken*	*Name of Bone*	*No. Taken*
Bone	5	Mandible	11
Humerus	6	Maxilla	6
Lumbar vertebræ	5	Skull	4
Femur	21	Scapula	4
Sternum	7	Rib	1
Ilium	7	Pubis	3
Sacrum	4	Clavicle	2
		Ischium	1
	Total 87		

Table V.—Aspiration Biopsies. Histological Diagnosis correlated with Site of Biopsy in Cases diagnosed as Malignant (368 Cases)

Adenocarcinoma (101)					
Breast	44	Humerus	1	Prostate	3
Gland	21	Thyroid	1	Bone	3
Scapula	1	Sternum	3	Kidney	2
Rectum	2	Abdomen	4	Chest wall	1
Sacrum	1	Intrathoracic	2	Ilium	1
Vertebra	1	Muscle	5	Pelvis	1
Palate	1	Site unknown	2	Skull	1
Sarcoma (33)					
Sacrum	1	Bone	3	Gland	3
Muscle	13	Maxilla	1	Pubis	1
Femur	3	Ilium	1	Retroperitoneal	1
Humerus	2	Mandible	3	Tibia	1
Thyroid Carcinoma (2)					
Thyroid	1	Skull	1		
Mixed Salivary Tumour (7)					
Gland	3	Parotid	4		
Carcinoma of Unknown Type (41)					
Breast	3	Muscle	2	Chest wall	2
?	6	Pelvis	1	Cheek	1
Thyroid	1	Gland	21	Orbit	1
Mandible	2	Liver	1		
Osteosarcoma (1)					
Bone	1				
Basal-cell Carcinoma (6)					
Cheek	3	Temple	1	Parotid	1
Bone	1				
Squamous Carcinoma (85)					
Sternum	2	Maxilla	3	Tongue	1
Gland	64	Site unknown	2	Ear	1
Penis	2	Vertebra	1	Chest wall	1
Mandible	4	Forehead	1	Fauces	1
Nose	1	Thyroid	1		
Melanocarcinoma (2)					
Skin	1	Muscle	1		
Hodgkin's Disease (4)					
Intrathoracic	1	Gland	2	Chest wall	1
Transitional-cell Epithelioma (21)					
Gland	15	Thyroid	1	Penis	1
Intrathoracic	1	Chest wall	2	Abdomen	1
Carcinoma of Lung (22)					
Intrathoracic	10	Chest wall	1	Sacrum	1
Gland	10				
Reticulum-cell Sarcoma (11)					
Gland	7	Pelvis	1	Abdomen	1
Sternum	1	Orbit	1		
Lymphosarcoma (1)					
Gland	1				
Chondrosarcoma (2)					
Bone	1	Sternum	1		
Plasma-cell Myeloma (2)					
Bone	2				
Type Unknown (22)					
Gland	7	Alveolus	1	Pelvis	3
Site unknown	1	Breast	1	Femur	1
Intrathoracic	1	Muscle	2	Bone (Paget's)	1
Soft tissue	1	Abdomen	1		
Prostate	1	Ischium	1		
Myeloblastic Myeloma (1)					
Perineum	1				
Fibrosarcoma (1)					
Gland	1				
Hypernephroma (1)					
Scapula	1				
Medulloblastoma (1)					
Lumbar vertebra	1				
Seminoma (1)					
Gland	1				

Aspiration biopsy, as can be seen from *Table I*, gave on the average about 63 per cent of useful results.

Drill Biopsy.—In 1944, having encountered a note by Christiansen in *Acta radiologica* of the possibility of drilling out a piece of tissue by means of a squared-off needle bevelled to have a cutting edge, I decided to try such a needle at the London Hospital with a dental drilling machine to provide the rotating power. The biopsies are referred to as ' drill biopsies '.

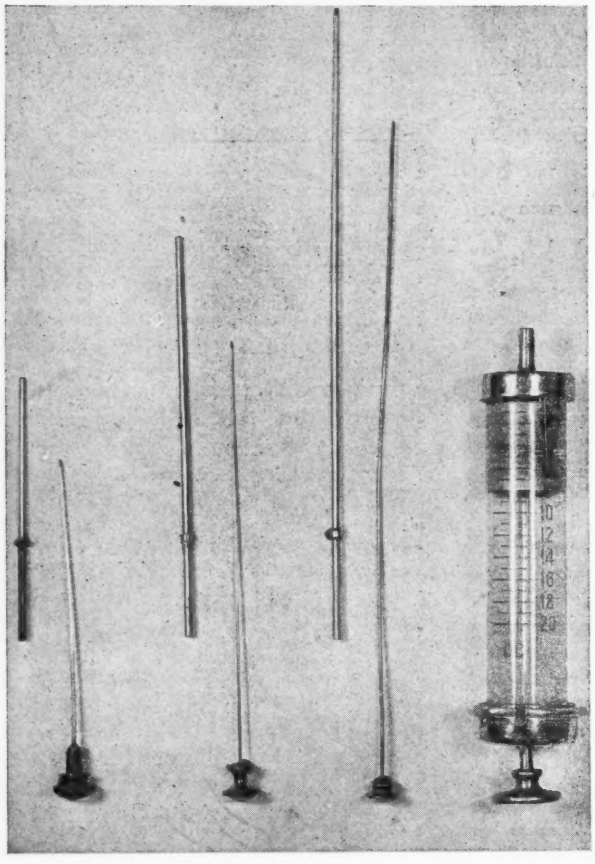


FIG. 339.—Squared-off bevelled needle for drill biopsy.

A survey of the results between October, 1944, and the end of February, 1946, is provided in *Table VI*. This should be compared with *Table II* giving a comparable survey of the results of aspiration biopsy. It is seen that the results of drill biopsy were consistently better than those of aspiration biopsy. This advantage is most marked in the case of biopsies taken from the breast.

These figures, moreover, do not represent all the advantages of drill biopsy because the pathologists' reports have been consistently more confident than those from the same pathologist of aspiration biopsy and the technique has been improved only recently, so that results are still better. At first, the needles used were ordinary needles with a bayonet fitting (for attachment to the syringe) which attached the needle securely during the drilling to an adaptor fitted into the driving mechanism of the drilling machine. Attached in this way it was difficult to get all the needles to rotate accurately on their axes. Therefore, needles, as shown in *Fig.* 339, were made in the workshops of the London Hospital. They have a similar sharpened end, but the attachment to the drilling machine obviates the use of an adaptor and the needles rotate more truly and better specimens are obtained. This improvement came into use during the last week in January, 1946.

Table VI.—DRILL BIOPSIES*

	NORMAL	DOUBTFUL	USELESS	BENIGN	MALIGNANT	TOTAL	PERCENTAGE OF USEFUL BIOPSIES
Bone	1	4	7	16	9	37	68
Glands	4	21	9	5	57	96	65
Liver	0	1	0	0	1	2	—
Thyroid	0	3	1	2	1	7	40
Intra-abdominal	0	2	0	1	7	10	80
Breast	1	1	0	1	18	21	90
Antrum	0	1	0	0	3	4	—
Other soft tissues	1	8	2	12	18	41	73
Totals	7	41	19	37	114	218	69

* This table does not include cases of carcinoma of prostate.

As complete a list as possible of the results of the drill biopsies carried out during the period mentioned is given in *Table VII*. Those referred to in *Table VI* as useful include only those reported as ' benign ' or ' malignant '.

EVALUATION OF NEEDLE BIOPSY

The Case for Needle Biopsy.—The foregoing tables indicate the usefulness of both aspiration biopsy and drill biopsy in the hands of the author, aided by the pathologists, but, as in all types of knowledge, figures, whatever they might prove, do not present or destroy the whole case.

In general, the type of case subjected to needle biopsy is one in which, because of lack of bed accommodation, lack of time, or lack of adequate indication in the view of the clinician concerned, a biopsy would not be taken in the ordinary way. In other words, much of the evidence provided for the pathologist by needle biopsies would not be available at all if they were not done. Moreover, the method is less dangerous in some ways than a biopsy by excision. The reason I first decided to try it was because the excision of a malignant cervical gland by a competent surgeon led to the rapid spread throughout the tissues of the neck and in the skin of the neck on to the chest wall, of cancer *en cuirasse*.

For the radiotherapist it is very important and much more satisfying to have a histological diagnosis not only of the condition from which the patient is suffering, but also of the lesion which he is treating. Moreover, it has been found by this method that the " sarcoma of bone " was a metastatic carcinoma, a gumma, or a pyogenic infection, that a " malignant gland of neck " was tuberculous, that a " carcinoma of œsophagus " was Hodgkin's disease, that a " retroperitoneal tumour " was a cellulitic spread from a diverticulitis, that a " mixed parotid tumour " for which operation had been refused was an adenolymphoma, and that a " lymphadenomatous gland " was a metastasis from a carcinoma of the bronchus. Such instances can be multiplied. Apart from these, the number of instances in which a clinical diagnosis is confirmed is sufficiently great, but there is a great number also in which this diagnosis is added to. Such cases are those in which a metastasis, by means of such a biopsy, may have the site of the primary growth defined or indicated for the first time. Again, there are cases in which the surgeon has not been able to make a diagnosis, sometimes even in spite of removing a gland at operation, in which the needle biopsy has provided a diagnosis of infection, reticulum-cell sarcoma, or metastatic carcinoma. In exposing a large gland an enlarged, but smaller, adjacent gland has been removed by eminent surgeons in two instances which I can call to mind, one in the neck and one in the abdomen, with in each case no histological result. The subsequent drill biopsy established a diagnosis in the first case of reticulum-cell sarcoma, probably originating in the suprarenal gland. Another useful feature of the method is that it often determines the presence of pus or of a cystic collection of fluid previously unsuspected.

The method of excising pathological lesions is doubtless, in general, more certain of providing information than is a needle biopsy. If needle biopsy fails to provide adequate evidence, however, surgical biopsy may still be resorted to. Methods of non-surgical treatment of cancer are being increasingly used and coincidently with this there is a tendency to dispense with histological evidence of the nature of the lesion. Results of such treatment, e.g., by œstrogens and radiation, apart from being misleading, will have no standing in international literature without histological evidence of their nature. Histology is still the touchstone of cancer work.

One way in which this method might be of value in radiotherapy and in chemotherapy, apart from verifying the diagnosis, is by facilitating the comparison of the states of the abnormal tissue at different times during treatment by taking serial biopsies.

One case description will help to emphasize the practical value of the method: A female patient had been ill for some months and I was asked to see her with a view to X-ray treatment of the lungs for what was thought by eminent physicians to be a slowly progressing infection of unknown nature, probably a fungus. I saw her at her home at 9 a.m. and, in the clinical examination, found a small gland 1 cm. in diameter deep behind the lower end of the left sternomastoid. She was very ill and I was averse to moving her the five miles to hospital, so I rang up the physician in charge of the case to ask permission to attempt an aspiration biopsy on the small gland. This obtained, I fetched the necessary apparatus and local anæsthetic and fortunately was able to obtain a small piece of the gland which was diagnosed at 11 a.m. the same morning by Dr. Hermitte as a metastasis of an oat-cell carcinoma of a bronchus.

This illustrates the ease with which such a biopsy can be effected, and in hospital the time spent is not usually more than three minutes from the time of starting to prepare the skin to the time of corking up the specimen tube. The patient's work or treatment need not be interrupted and delay because of bed difficulties is unnecessary.

The Case against Needle Biopsies.—From the pathologist's point of view it is better to have a big specimen than a small one, and, indeed, pathology and clinical medicine would be less advanced than they are if surgery had not developed as it has. From the patient's point of view, however, the more conservative the treatment the better, provided the effects and results are the same.

There is no doubt that these biopsies are useless if the pathologist is not patient. Especially is patience needed during his first attempts to provide diagnoses from pieces smaller than those he has been used to discarding. The first aspirations having been successful, the next few biopsies attempted were more ambitious and the pathologist wrote: " I think the practice should be discontinued ". However, he was persuaded to persevere and became enthusiastic. The results obtained since with aspiration biopsy, and to a greater extent by drill biopsy, indicate the usefulness of the method. It must be used with judgement, however, and not as the method of choice in cases more suitable for ordinary biopsies. For instance, a bronchoscopy should be the method of choice to obtain a biopsy from a bronchial carcinoma, but if this fails a drill biopsy may be successful. But if the drill biopsy provides histology which would otherwise be lacking or can save the patient from a major operation, it is justifiable even if the pathologist receives only a small piece of tissue.

TYPES OF DRILL BIOPSY

The types of drill biopsy carried out were :—

1. Relatively Superficial Soft-tissue Lesions.—These include cervical, axillary, inguinal, and other lymph-nodes, breast lesions, muscle lesions, and lesions in such situations as the thyroid gland, parotid or other salivary glands, and the orbit. Also regions such as the tonsil and tongue have been drilled when the site of non-ulcerated lesions. Local anæsthesia is easy in such cases, and the procedure is safe and simple.

2. Easily Accessible Bony Lesions.—These include lesions in all the bones except the vertebræ. The bone is inspected and carefully measured on the radiographs to determine the site of entry to be attempted by the drill, aiming at an active part of the pathological process and ease of approach. If there is an obvious swelling it is very easy of course, but if not, it is sometimes possible to find a point where the cortical bone is thin or destroyed and the needle can easily be pushed into the lesion. If the

Table VII.—DRILL BIOPSY REPORTS

No.	Clinical Diagnosis	Biopsies		Drill Biopsies							Primary	Remarks
		Other Sites	Same Site	Site	Normal	Doubtful	Useless	Benign	Malignant	Type		
	Breast :—											
1381	Carcinoma	..	..	Breast	..	..	..	..	+	Polygonal-cell carcinoma	Breast	..
O/217	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
162	Ca.	..	..	Breast (1)	..	..	..	+	..	Abscess	..	..
				Breast (2)	..	..	..	..	+	Scirrhous ca.	Breast	..
286	Recurrent ca.	..	..	Nodule in scar	..	..	..	..	+	Poly.-cell. ca.	Breast	..
O/54	Rec. ca.	..	..	Nodule in scar	..	..	..	..	+	Poly.-cell. ca.	Breast	..
A/146	? Ca.	..	..	Breast	+	..	..	..	..	..	..	..
A/637	Ca. R.	L. breast (−) L. axilla (−)	..	R. breast	..	..	..	..	+	Scirrhous ca.	Breast	..
A/695	Ca. R.	L. breast (−)	..	R. breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
A/497	Ca.	Ovarian tumour (−)	..	L. breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
A/869	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
1422	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
A/380	Ca.	..	..	Breast	..	..	..	..	+	Spheroidal-cell. ca.	Breast	..
144	Rec. scar.	..	..	Chest wall	..	+	..	..	..	..	..	..
B/28	Ca.	..	..	Breast	..	..	..	..	+	Trabecular poly.-cell. ca.	Breast	..
A/695	Ca.	..	..	Breast	..	..	..	..	+	Trab. poly.-cell. ca.	Breast	..
A/1176	Ca.	..	..	Breast	..	..	..	..	+	Trab. poly.-cell. ca.	Breast	..
B/19	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
A/562	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
162	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
A/439	Ca.	..	..	Breast	..	..	..	..	+	Poly.-cell. ca.	Breast	..
	Bone :—											
1424	? Hypernephroma ? Osteoclastoma	..	..	Sacrum	..	..	..	..	+	Myeloma	..	..
121	Ca. breast	..	..	Tibia	..	..	..	..	+	Poly.-cell ca.	..	..
815	? Angioma ? Metastasis from melanoma	Forearm (− melanoma)	..	1st L.V.	..	..	+	..	..	..	..	Diagnosis later, duodenal ulcer
1523	Osteoclastoma	..	..	Mandible	..	..	..	+	..	Osteoclastoma	..	..
A/2	Metas. from prostate	..	..	Iliac bone	..	..	..	..	+	Poly.-cell-like squamous cells	..	..
A/811	? Osteoclastoma	..	..	Iliac bone	..	..	..	+	..	Colliquative necrosis	..	..
A/649	Secondary ca. femur primary ? hypernephroma	..	..	Upper end of femur	..	..	..	..	+	Myeloma	..	..
A/634	Osteoclastoma	..	..	Os calcis	..	..	..	+	..	Osteoclastoma	..	..
A/612	Osteoclastoma	..	..	Iliac bone	..	+	..	..	..	..	..	..
A/567	Bronchial neoplasm ? metas.	..	..	Rib	..	..	..	..	+	Poly.-cell ca.	..	..
A/839	Sacral tumour	..	..	Sacrum (1)	..	..	+	..	..	..	Diagnosis unknown	Due to artefact
				Sacrum (2)	..	+	..	..	..	? Tubercle		
				Sacrum (3)	..	..	+	..	..	..		
A/862	? Osteoclastoma	..	..	Radius	..	..	..	+	..	Osteomyelitis	..	..
A/1080	Osteoclastoma	..	..	Jaw	..	..	..	+	..	Osteoclastoma	..	..
A/1065	Chondroma ? malignant	..	..	Tumour of ilium	..	..	..	+	..	Chondroma	..	..
A/340	?	..	..	Shoulder	..	+	..	..	..	? Inflam.	..	..
				Shoulder	..	..	..	+	..	Purulent granulation tissue	..	23 days' interval
B/74	? Sarcoma femur	..	..	Femur	..	..	..	+	..	Pyogenic gran. tissue	..	..
B/31	Pathological fracture clavicle	..	..	Clavicle	..	..	..	..	+	Ca.	..	Unknown primary
A/371	Osteoclastoma tibia	..	..	1. Tibia	..	..	..	+	..	Osteoclastoma	..	..
				2. Tibia	..	..	..	..	+	Malignant osteoclastoma	..	..
A/962	Unknown lesion tibia	..	..	Tibia	..	+	..	..	..	Structureless jelly	..	..
B/122	Ca. cervix	Cervix ca.	..	Pubis	..	..	..	..	+	Small cells malig. Not like ca. cervix	..	..
A/1207	Ca. bladder ? metas. ? myelomatous	Bladder ca. 15 yr. prev.	..	Clavicle	..	..	+	..	..	Muscle only	..	..
B/7	Unknown lesion terminal phalanx	..	..	Terminal phal-								

A/1207	Ca. bladder ? metas. ? myelomatous	Bladder ca. 15 yr. prev.	..	Clavicle	..	..	..	..	..	..	..	..
B/7	Unknown lesion terminal phalanx	..	..	Terminal phalanx finger	..	..	+	..	..	Blood-clot only	..	..
A/494	Lesion femur	..	..	Femur	..	..	..	+	..	Chronic inflam.	..	..
A/387	Tumour rib	..	..	Rib	..	..	..	..	+	Sec. ca.	? Lung	..
1307	? Osteoclastoma humerus	..	..	Humerus	..	..	..	+	..	Osteoclastoma	..	..
A/601	Fracture femur	..	..	Femur (neck)	+	..	..	..	..	Bone with osteoclasts	..	Subsequent diagnosis : fracture —no lesion
634	Chondroma femur	..	..	Femur	..	..	..	+	..	Chondroma	..	Not malignant yet
A/1080	Tumour mandible	..	..	Mandible	..	..	..	+	..	Osteoclastoma	..	..
A/1065	Chondroma ilium	..	..	Ilium	..	..	..	+	..	Chondroma	..	..
A/1029	Tumour humerus	..	..	Humerus	..	..	..	+	..	Osteoclastoma	..	..
A/765	? Sarcoma tibia	..	..	Tibia	..	..	..	+	..	Caseous granuloma	? Gumma	W.R. (– –)
A/726	? Ca. vertebra	..	..	L. V.	..	..	+	..	..	Fat and marrow	..	..
A/630	? Sacral tumour	..	..	Sacrum	..	..	+	..	..	..	..	..
	Glands of Neck :—											
A/535	? Tubercle	..	..	Neck	..	..	+	..	..	..	..	..
A/4	Ca. bronchus	..	..	Cervical gland	..	..	..	..	+	Scirrhous squamous	..	..
A/430	Hodgkin's disease	Gland excised (–)	..	Left supraclavicular	..	+	..	..	..	Suggests Hodgkin's	..	Fibrous tissue, one cellular bit showing numerous oesinophil leucoyctes and large mononuclear. ? Reticulum cells but no giant cells
A/576	Squamous epithelioma arising from mandible	..	..	Cervical gland	..	..	..	..	+	Sq. ca.	..	..
A/404	Ca. pinna	Below ear (–) unlike a primary ca.	..	S.C.	..	..	..	..	+	Spheroidal-cell ca.	? Bronchus	..
A/653		Gland excised L. sub. (–)	..	L. submax. region	..	..	..	..	+	Mixed tumour	..	..
1280	Ep. L. ear with metastases	..	..	Neck	..	..	..	..	+	Sq.-cell ca.	..	..
A/441	Ca. bronchus	..	..	1. S.C.	..	..	+	..	..	..	Unknown	..
				2. Pretracheal mass	..	..	+	..	..	..	Unknown	..
A/403	Ca. breast ? metas.	Breast (–)	..	L.S.C.	..	..	+	..	..	..	Breast	Nature of drilled lesion uncertain
A/204	Ovarian ca.	..	..	L.S.C.	..	..	..	..	+	Poly.-cell ca.	? Dysgerminoma ovary	..
A/271	Ca. pyriform fossa	Pyriform fossa (–) R.U.	..	R. neck	..	..	+	..	..	..	Basal-cell ca. neck	Gland biopsy—necrosed tissue
A/92	Sec. ca. gland neck ? primary	..	..	Neck	..	..	..	..	+	Sq.-cell ca.	..	..
A/16	Ca. tongue submax. tumour	..	..	Submax. gland	+	..	..	..	..	..	..	..
1432	Ca. lung	..	Aspiration (useless)	L.S.C. gland	..	..	..	..	+	Poly.-cell ca.	Bronchus	..
1350	Hodgkin's	..	..	Cervical gland	..	..	..	..	+	Lymphosarcoma	..	..
1396	? Hodgkin's	..	..	Cerv. gl.	..	+	..	..	..	Probably Hodgkin's	..	..
1071	Dysgerminoma	..	..	S.C.	..	..	..	..	+	Large spher.-cell ca.	..	Probably sec. ca.
1436	Lymphadenoma	Post. nares (–)	..	L. neck	..	..	+	..	..	..	Lymphadenoma	..
1490	? Bronchus	Gland excision (–)	..	Cervical	..	..	..	..	+	Poly.-cell ca.	?	..
1368	Antral tumour	Section (– Ewing's)	..	R. submax.	..	..	..	..	+	?	?	..
1511	Ep. palate	Palate (–)	..	Gland of neck	..	..	..	..	+	Sq.-cell ca.	..	..
A/921	? Hodgkin's ? metas. neck glands	..	..	S.C.	..	..	..	..	+	Hodgkin's	..	..
	Glands :—											
A/1035	Ca. tongue	..	..	Gland neck	..	..	..	..	+	Sq.-cell ca.	Sq. ep.	..
A/450	Ca. epiglottis	..	..	Neck	..	..	..	..	+	Ca.	?	..
A/1079	Ca. alveolus	..	..	Neck	..	..	..	..	+	Ca.	Sq. ep.	..
A/306	Ca. floor of mouth	..	..	Neck (after)	..	+	..	..	..	Necrosed tissue	..	Necrosed tissue
A/1250	Leukæmia (Ly)	..	..	Neck (after)	..	+	..	..	..	Fib. gran. tissue	..	..
A/1118	Ca. breast	Breast	..	Neck	..	..	..	..	+	Trab. poly.-cell ca.	..	..
B/88	Ca. lung	Bronchus	..	Neck	..	+	..	..	..	Fibroblastic poly.	.	..

Table VII.—DRILL BIOPSY REPORTS—*continued*

No.	Clinical Diagnosis	Biopsies		Drill Biopsies							Primary	Remarks
		Other Sites	Same Site	Site	Normal	Doubtful	Useless	Benign	Malignant	Type		
1154	Ca. thoracic dermoid ? Metas.	Mediastinum	..	Axilla	..	+	..	..	..	Necrosed tissues	..	..
B/10	Hodgkin's	..	Neck doubtful	Neck	..	..	..	..	+	? Reticulum-cell sarcoma	..	Better than surgical biop.
A/1046	Retroperitoneal tumour	Anaplastic tu. (retrop.)	..	Groin	..	+	..	..	..	Fibrous tissue	..	..
A/1127	? Hodgkin's	Cerv. gland lymphoid follic. retic.	..	Groin	..	+	..	..	..	? Lymphoid follic. retic.	..	Diagnosis not possible on drill biopsy
A/1044	Ca. cervix	Cervix adenoca.	..	Groin	..	..	..	..	+	Tubular-columnar cell ca.	..	..
B/29	? Ca. lung	..	..	Neck	..	..	..	..	+	Poly.-cell ca.	..	..
O/80	Ca. breast	Breast ca.	..	Axilla	..	..	..	..	+	Poly.-cell ca.	..	..
B/4	? Ca. kidney	..	..	Groin	..	..	..	..	+	Poly.-cell ca.	? Testis or adrenal cortex	..
A/1255	Ep. ear	Ear ep.	..	Neck	..	+	..	..	..	Chr. inflam.	..	..
A/1195	Tb. gland	..	..	Neck	..	..	..	+	..	Tuberculosis	..	..
A/1142	? Reticulosis	..	..	Neck	..	..	..	..	+	Retic. sar. or Hodgkin's	..	..
A/734	? Sc. metas. (breast)	Breast ca.	..	Neck	+	..	..	..	..	Fat only	..	..
A/721	Ca. tongue	..	..	Neck	..	..	..	..	+	Well diff. sq. ca.	..	..
A/935	? Tuberculosis	..	..	1. Neck	..	+	..	..	..	Chr. inflam. with granuloma	..	..
				2. Axilla	..	..	..	..	+	Hodgkin's	..	..
A/1202	? Tb. gland neck	..	..	Neck	..	+	..	..	..	Blood and neutrophil leucocytes	..	..
A/1208	Ca. epiglottic fold	..	..	Cerv. gland	..	..	..	..	+	Poly. colum. tubular ca.	..	..
A/435	Ca. tonsil	..	..	1. Neck	..	..	..	..	+	Necrotic sq. cell ca.	..	During tr.
			..	2. Neck	..	..	..	..	+	Ghosts of poly.-cell ca.	..	3/12 after tr.
A/1263	Ca. tongue	Tongue—(1) Failed (2) Ca.	..	Neck	..	..	..	..	+	Poorly diff. sq. ca.	..	..
A/1138	Hodgkin's	..	..	Neck	..	..	..	..	+	Poly.-cell ca. pharynx or lung	..	..
A/1086	? Ca. in gland and tubercle	..	1. Tubercle 2. Carcinoma	Neck	..	..	..	..	+	Sq. cells.	..	..
A/423	Malig. melanoma foot	Foot malig. melanoma	..	Groin	..	..	..	..	+	Malig. melanoma		..
A/535	? Ca. metas. in gland	..	..	Neck	..	+	..	..	..	Chr. inflam.	..	..
A/354	Ca. tongue	Tongue sq. ca.	..	Neck	..	..	..	..	+	Sq.-cell ca.	..	..
1384	Ca. nasopharynx	..	..	Axilla	..	..	..	..	+	Sq.-cell ca.	..	..
1431	? Hodgkin's. ? Ca. bronchus	..	..	Axilla	..	..	..	..	+	Poly.-cell ca.	? Bronchus	..
1371	Ep. anus	..	..	Groin	+	..	..	..	..	..	..	..
A/322	? Ca. ovary. ? Metas. ca. ? Dermoid	..	..	Axilla	..	..	..	..	+	Sph.- or poly.-cell ca.	? Dermoid cyst	..
1529	Cerebral tumour	..	..	Neck	..	..	..	..	+	? Lymphosarcoma	..	..
A/159	Ca. tongue	Tongue sq. ca.	..	Neck	..	..	..	..	+	Sq. ca.	..	..
962	Ca. lung	..	..	Neck	..	..	..	..	+	Small poly.-cell sq. ca.	? Bronchus	..
A/67	Mediastinal tumour	..	..	Neck	..	..	..	..	+	Oat.-celled ca.	Bronchus	..
A/529	Reticulosis	..	..	Axilla	..	..	..	..	+	Ret. sar.	..	..
A/228	Ca. lip	Lip sq. ca.	..	Submax.	..	..	..	..	+	Sq.-cell ca.	..	..
A/956	Ca. breast	Breast ca.	..	Neck	..	..	+	..	..	Vol. muscle and nerve	..	..
A/909	Ca. œsophagus	..	..	Neck	..	..	..	..	+	Hodgkin's dis.	..	..
A/861	Ca. larynx	..	..	Neck	..	..	..	..	+	Sq. ca.	..	..
A/663	Mass in neck	..	..	Neck	..	..	..	..	+	Sq. ca.	Unknown	No primary yet found
1252	Ca. fauces	..	..	Neck	..	..	..	..	+	Sq. ca.		
O/33	1. Inguinal gland	..	Adenocarcinoma (4 yr. before)	Groin	..	..	..	..	+	Adenoca.	Uterus ovary	Reviewed
	2. Inguinal gland	..	Adeno-carcinoma (4 yr. before)	Groin	..	..	..	+	..	Endometriosis	..	Decided endometriosis. Subsequent development suggests malignant disease

458	Ca. breast	Breast ca.	..	Neck	..	..	..	+	..	Fibrous tissue	..	..
825	1. Ca. larynx	Larynx ca.	..	Neck	..	+	..	..	..	Chr. inflam.	..	..
	2. Ca. larynx	Larynx ca.	..	Neck	..	..	..	..	+	Sq. ca.	..	..
657	1. ? Ca. larynx	Larynx ca.	..	Neck	..	..	+	..	..	..	..	Diagnosis could not be decided for many months
	2. ? Ca. larynx	Larynx ca.	..	Neck	..	+	..	..	..	Chr. inflam.	..	Died ca. larynx
A/392	? Ca. larynx	..	..	Neck	..	+	..	..	..	Chr. inflam.	..	..
	Glands of Neck :—											
O/10	Ca. breast. ? Metas.	Breast (−)	..	S.C. gland	..	..	..	..	+	Poly.-cell ca.	Breast	..
327	Lympho-epithelioma	..	..	Cerv. gl.	..	..	..	..	+	Poly-.cell ca.	? Derived from a sq. ca.	..
118	Lympho-epithelioma	Nasopharynx (−) (Lympho-ep.)	..	Cerv. gl.	..	..	..	..	+	Poly.-cell ca.	? Lung	..
A/1104	Sec. ca. Primary unknown	..	..	Neck	..	..	..	+	..	Tubercle	Gl. of neck	..
A/1073	Ca. tonsil sec. in neck	..	..	R. neck	..	..	..	..	+	Hodgkin's	..	..
A/935	? Ret.-cell sarcoma	Axilla (drill —)	..	R.S.C.	..	+	..	..	..	? Chr. inflam. ? gumma	Hodgkin's proved by drill biop. from neck	..
A/1034	Tb. adenitis	Gland excised (−)	..	L. neck	..	..	..	..	+	Hodgkin's	..	..
A/450	Ca. larynx	..	..	R. neck	..	+	..	..	..	..	..	3 Drill biopsies: 2, 3 — pus and necrosed tissue. Probably sec. ca.
A/792	? Nasopharynx ca. Metas. neck	..	..	L. neck	..	+	..	..	..	Chr. inflam. (after deep X-ray)	Diagnosis unknown	..
A/979	Tumour behind L. ear. ? Metas. neck	Ear (inflam.)	..	R. neck	+	..	..	..	..	..	..	..
1519	? Ca. antrum	..	..	Neck	..	+	..	..	..	? Abscess	..	..
				Neck	..	+	..	..	..	? Hodgkin's	..	..
A/392	Ca. larynx	..	..	Neck	..	+	..	..	..	..	Diagnosis not certain	..
B/10	Hodgkin's	Exc. of gl.(doubtful)	..	R. neck	..	..	..	..	+	Hodgkin's	..	..
A/137	Malig. glands neck	..	..	R. neck	..	..	..	..	+	Sq.-cell ca.	? Bronchus ? Nose or pharynx	..
A/590		..	..	..	..	..	..	+	..	Tuberculosis	..	..
A/663	1. Gland of neck	..	..	R. neck	..	..	+	..	..	..	..	Primary
	2. Unknown primary	..	..	R. neck	..	..	..	..	+	Sq. ca.	?	unknown
	Liver :—											
1490	Ca. bronchus	Sec. glands of neck	..	Liver	..	..	..	..	+	Sec. ca.	? Lung	..
A/547	? Ca. pancreas with metas.	..	..	Liver	..	+	..	..	..	..	..	..
	Thyroid :—											
A/29	? Ca. larynx	..	..	Thyroid cart.	..	+	..	..	..	..	..	..
A/5	Ca. thyroid	..	..	Thyroid	..	+	..	..	..	..	Not certainly ca.	Prob. ca.
278	Ca. thyroid	..	Asp. biop. (useless)	Thyroid	..	+	..	..	..	..	Diagnosis unknown	..
A/988	Ca. thyroid	..	..	Thyroid	..	+	..	..	..	..	Ca. thyroid	..
A/215	Ca. thyroid	..	..	R. lobe thyroid	..	..	..	..	+	Poly.-cell ca.	Thyroid	..
A/1211	Ca. thyroid	..	..	Thyroid	..	..	..	+	..	Riedel's goitre	..	..
A/365	Thyroid tumour	..	..	Thyroid	..	..	..	+	..	Adenoma or diff. goitre	..	..
A/447	Riedel's thyroiditis	..	..	Thyroid	..	..	+	..	..	..	..	..
	Intra-abdominal :—											
A/866	? Ewing's ? Neuroblastoma	..	..	L. abdomen	..	..	..	..	+	Poly.-cell tu., possibly ca.	..	..
A/818	Neurofibroma	..	..	1. R. upper abdomen	..	..	..	..	+	Neurofibroma	Neurofibromatosis	..
				2. L. iliac tu.	..	..	..	..	+	Neurofibroma		..
A/831	? Ca. ovary ? Dysgerminoma	..	..	Umbilicus	..	+	..	..	..	..	..	..
A/101	? Arising from prostate	Gland at operation (useless)	..	Lower abdom. tumour	..	..	..	..	+	Large poly. cells	Ca. cortex of suprarenal	..
1321	Epigastric and pelvic tumours	Skin of abdom. wall	..	Upper abdom. tu.	..	..	..	..	+	Small poly. cell	?	..

Table VII.—DRILL BIOPSY REPORTS—*continued*

No.	Clinical Diagnosis	Biopsies		Drill Biopsies						Type	Primary	Remarks
		Other Sites	Same Site	Site	Normal	Doubtful	Useless	Benign	Malignant			
1482	Endometriosis	..	..	Umbilicus	..	..	..	+	..	Endometrioma	..	..
A/427	Tumour lower abdom.	..	..	Lower abdom.	..	+	..	..	..	..	..	? Ca. ovary
A/108	Ca. testis	..	..	Abdom. tu.	..	..	..	..	+	Poly.-cell ca.	? Suprarenal cortex	..
A/320	? Ca. bladder ? Ca. prostate	..	..	Pelvic mass	..	..	..	..	+	Sq. ep.	..	..
	Antrum :—											
1343	Ca. L. antrum	..	..	L. antrum	..	+	..	..	..	Necrosed tissue	Diagnosis not certain	..
A/557	Ca. antrum	..	..	Antrum	..	..	..	..	+	Large poly.-like sq. cell	..	..
A/857	Ca. antrum	..	..	Antrum	..	..	..	..	+	Sq.-cell ca.	..	..
A/820	Ca. antrum	..	..	Antrum	..	..	..	..	+	Sq. ca.	..	
	Intrathoracic :—											
A/1011	Ca. lung	..	..	Lung	..	..	..	..	+	Poly.-cell ca.	? Lung	..
A/929	Ca. lung	..	..	Lung	..	..	..	..	+	? Ca. ? Endothelioma	..	..
A/528	Sarcoma chest wall. ? Metas. lung	..	..	Lung	..	..	..	..	+	Seromyosarcoma or fibrosarcoma	..	..
*B/269	Ca. bronchus	..		Lung	..	..	..	..	+	Spheroidal and spindle-celled ca.	Thymus	..
*B/79	Chondro-osteo-sarcoma	..	..	Lung	..	..	..	..	+	Mostly fibrous tissue with numerous trabeculæ of imp. bone and osteoid tissue; little chondroid tissue. One tube lined with flattened and cubical epithelial cells in middle	Jaw	..
*B/379	Ca. lung	..	..	Lung	..	..	+	..	..	Purulent necrosed lung tissue containing concentrically laminated necrosed nodules. No neoplastic tissue.	..	..
*B/551	? Ca. lung	..	..	Lung	..	..	+	..	..	Pieces of tissue-like normal liver	..	..
*B/341	Ca. lung	..	..	Lung	..	..	..	..	+	Sq. celled ca.	..	..
*B/227	Ca. bronchus	..	..	Lung	..	..	..	..	+	Polygonal cells and tubules of columnar cells. Very fine specimen	? Lung or bronchus	..
	Prostate :—											
*161	All these cases were under treatment by stillbœstrol	..	..	..	..	..	+	..	..	Voluntary muscle and fibrous tissue. No prostatic tissue	..	..
*162		..	..	..	..	+	..	..	..	Possibly myo-adenoma of prostate. No. ca.	..	..
*163		..	..	..	..	..	..	..	+	Col. tubular poly.-cell ca. prostate	..	..
*164		..	..	..	..	..	+	..	..	Involuntary muscle. No acini. No abnormality	..	..
*165		..	..	..	..	+	..	..	..	Fibrous tissue with involuntary muscle-fibres. No acini	..	..
*166		..	..	..	..	+	..	..	..	Suggestion of chronic inflammation of fibrous tissue	..	..
*B/594	..	..	..	..	..	..	..	..	..	Col. tubular poly.-cell prostate	..	..
*A/1175	..	..	..	..	..	..	..	..	+	Solid polygonal-celled ca.	..	..
	Other Soft Tissues :—											

	Other Soft Tissues :–											
B/12	Mixed parotid tumour	..	..	R. parotid	..	..	..	+	..	Adenolymphoma	R. parotid	..
A/1094	Metas. glands groin	Cervix (–), Tumour of leg (?)	..	R.I.F.	..	..	..	..	+	Sec. tubular columnar-cell ca.	? Large gut	..
1278	Rec. hydronephroma	Excision (–)	..	Tumour nr. scar	..	..	..	..	+	Grawitz	Kidney	..
A/71	Rec. ca.	Breast ca.	..	Tumour nr. sternum	..	..	..	..	+	Poly.-cell ca.	..	..
A/909	Ca. œsophagus	..	..	Tumour of neck	..	..	..	..	+	Hodgkin's	..	..
A/1168	Tumour tonsil	Tonsil. Mixed salivary-gland tumour	..	Mass in neck	..	..	..	Gland tumour	..	Mixed salivary gland type	..	..
A/984	Tumour tonsil	Lymphatic leukæmia	..	Tonsil	..	..	..	+	..	Chr. inflam.	..	Cleared up
B/85	Tumour buttock	..	..	Buttock	..	..	..	..	+	Ca.	Unknown	..
A/463	Rec. ca. breast	Breast ca.	..	Chest wall	..	..	..	+	..	Fat only	..	..
A/1194	Swelling in neck. ? Metas. from thyroid	..	..	? Tumour of neck	..	+	..	..	..	Fat only	..	..
B/109	Mass in chest wall. ? Hodgkin's	..	..	Chest wall	..	+	..	..	..	Fibrotic tissue	..	..
B/195	Tumour in neck. ? Ewing's. ? Neuroblastoma of adrenal	..	..	Mass in neck	..	..	+	..	..	Blood-clot and vol. muscle only	..	..
B/123	Malig. melanoma finger	Finger malig. melanoma	..	Thickening in axillary scar	..	..	..	+	..	Fibrous tissue	..	..
A/29	Ca. larynx	..	..	Thyroid cartilage	..	+	..	..	..	..	..	..
A/993	Ca. cæcum	..	..	Chest wall	..	..	..	..	+	Trab. ca.	..	..
810	? Lipoma chest wall ? Rec. ca.	Breast ca.	..	Chest wall	..	..	..	+	..	Lipoma	..	..
A/827	? Rec. ca. ? Fibrosis	Lip. ca.	..	Nodule in lip	..	..	..	+	..	Fibrous tissue	..	..
A/845	Tumour of thigh	..	..	Thigh	..	..	..	..	+	Fibrosarcoma	..	..
A/687	Tumour of leg	..	..	Leg	..	..	..	..	+	Ewing's tumour	..	..
O/32	? Rec. ca. breast	Breast ca.	..	Chest wall	..	..	..	..	+	Poly.-cell ca.	Breast	..
1516	? Reticulosis	..	..	Tumour of skull	..	..	..	..	+	Myelomatosis	..	..
1501	Tumour vertebra	..	..	Tumour of vertebra	..	+	..	..	..	..	Chondroma of vertebra	Tumour in op. scar. Diagnosis not certain
25	? Fibrosarcoma thigh	..	Aspiration doubtful	Tumour thigh	..	..	..	..	..	Angio-endothelioma	..	..
A/124	? Sarcoma	..	..	Tumour thigh	..	+	..	..	..	..	..	..
A/533	Mediastinal neoplasm	..	..	Tumour axilla	..	..	+	..	..	..	..	..
A/704	? Myosarcoma	..	..	Tumour fixed to humerus	..	..	..	+	..	Fibrotic granulation tissue	..	..
A/720	Fibrosarcoma	..	..	Medial aspect of thigh	..	..	..	..	+	Poly.-cell ca.	Grawitz's ca.	..
A/784	? Sarcoma. ? Abscess	..	..	Thigh	..	..	..	+	..	Abscess	..	..
A/670	? Rec. fibrosarcoma	Excision 1941 (–)	..	Thigh	..	+	..	..	..	..	Diagnosis of lump drilled is uncertain	..
A/499	Parotid tumour	..	..	Parotid	..	..	..	+	..	Adenoma of salivary gland	..	..
A/902	? Ossifying chondroma	..	..	Tumour in muscle	..	+	..	..	..	..	..	..
A/396	Ca. larynx	..	..	Extra-laryngeal tissue	..	+	..	..	..	..	..	Drilled after deep X-ray treatment
A/316	? Fibrosarcoma	..	..	R. thigh	..	..	..	..	+	Fibromyosarcoma	..	..
1265	Ca. breast. ? Metastases	..	..	Tumour fixed to rib	..	..	..	..	+	Poly.-cell ca.	Breast	..
A/57	Fibrosarcoma	..	..	L. cheek	..	..	..	..	+	Fibrosarcoma	..	..
1520	Actinomycosis	..	..	Tonsillar region	..	..	..	+	..	Chr. inflam. No actino.	..	..
A/35	Ep. L. pinna	..	..	Post-auricular	..	..	..	..	+	Poly.-cell ca.	..	..
1313	Ca. breast. ? Metastases	..	..	Chest wall	..	..	..	..	+	Secondary ca.	Breast	..
A/959	Ca. lip. Metas. mandible	Lip (–)	..	Lower chest	..	..	..	..	+	Sq. cell	L. submax.	..
A/152	Ca. breast. ? Metas. chest wall	Breast (–)	..	Tu. fixed to rib	+	..	..	..	..	..	..	..

Those cases marked with an asterisk are more recent than the original specimens on which the tables for this article have been based.

cortical bone is dense I drill a hole in it with a bone drill worked by hand and then push the hollow needle through the same hole into the lesion.

3. The Vertebræ.—

The *cervical vertebræ* almost belong to the last group, but obvious precautions are taken to avoid the vertebral artery, the spinal cord, and nerve-roots.

The *dorsal and lumbar vertebræ* are more difficult. The neural arches are easily accessible but not often affected. The bodies are deeply situated. radiographs were made with a portable machine, correction may be necessary for the divergence of the beam in determining the distance from the midline of the point to be aimed at. The lesion usually feels firmer than the lung through which the drill is passed. When the firmer tissue is reached, the trocar is removed and drilling commenced. The heart, aorta, and superior vena cava are all, of course, structures to avoid, but with rational confidence in one's judgement, accidents do not occur,

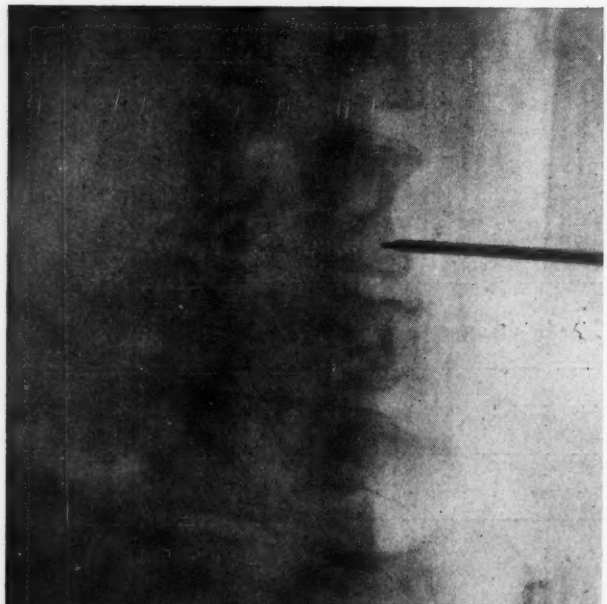


FIG. 340.—Needle incorrectly placed.

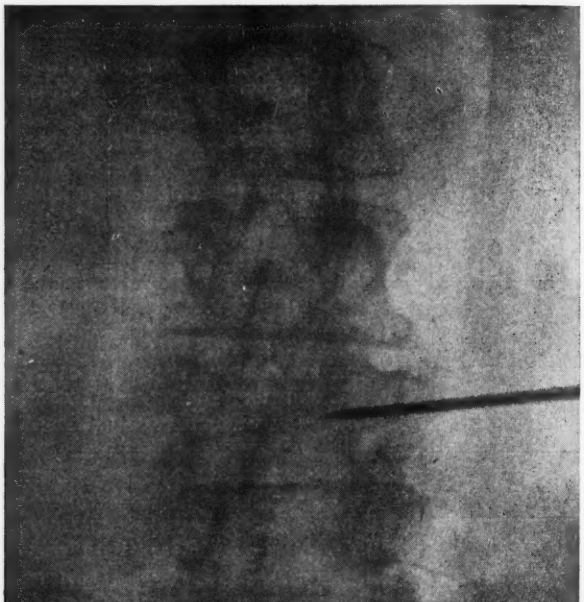


FIG. 341.—Needle correctly placed.

From lateral radiographs taken with a marker on the skin in the midline, the depth (6–8 cm. usually) of the lesion from the skin can be measured. If the drill is then inserted through the skin at an equal distance from the midline at an angle of 45° to the vertical with the patient lying prone, it is not difficult to strike the lesion. This procedure is best carried out under light general anæsthesia and with facilities for radiographic verification of the position of the drill. Pentothal is adequate with premedication and efficient radiography (*see Figs.* 340, 341). A grid used on the back for an anteroposterior radiograph with the patient supine helps to decide on the level to insert the drill through the skin (*Fig.* 342).

4. Intrathoracic Lesions.—In a radiograph the ribs form adequate markers for the chest, and by drawing lines from front to back it is possible to determine the direction in which the drill should be passed to the lesion. The patient should be in the same position as when the radiograph was taken. The distance from the midline is easy to determine, but in some cases, especially if the although the proximity of the heart or aorta may cause alarming excursions of the free end of the drilling needle. Local anæsthesia is often adequate, but a general anæsthetic more comfortable.

5. Intra-abdominal Lesions.—These cause most trepidation unless there is a large tumour reaching the surface. It is necessary to judge if the tumour is retroperitoneal, in which case it may be reached through the back, or, in the case of a pelvic tumour, extraperitoneally by passing the needle below Poupart's ligament into the tumour.

The tumour may sometimes reach the surface of the abdomen over only a small area. It is possible sometimes to push the bowel out of the way. Only when sure that the tumour can be punctured without the risk of going through bowel should the drill be inserted. In this way a biopsy can be obtained without difficulty with novocain anæsthesia in many patients. In one patient with a tumour between the liver and the kidney I adjudged it safest to penetrate the tumour by going through the liver, the resultant biopsy changing the diagnosis from Hodgkin's

disease to neurofibrosarcoma. In another case a retroperitoneal tumour sent for radiotherapy proved to be a hydatid cyst.

6. Prostatic Lesions.—These are best dealt with in the lithotomy position. It is necessary to have the left index finger in the rectum. The needle is inserted through a small stab in the skin of the perineum, with the patient anæsthetized (preferably general). The needle is easily felt, and can be guided into the prostate. The trocar is then removed and drilling commenced.

TECHNIQUE

The techniques of the methods employed in this series of cases are different for aspiration biopsy and drill biopsy from the point of view of obtaining the tissue, and there are slight differences in the treatment by the pathologist of the material obtained.

Aspiration Biopsy.—The technique for obtaining the tissue is the same as that described by Martin and Ellis (1930).

A sharp hollow needle with an internal bore of about 1·5 mm. diameter is attached to a 20-c.c. syringe with a closely fitting piston. The tumour is fixed, if necessary, by the left thumb and index finger (e.g., in the case of an accessible tumour such as a lymph-node, breast tumour, or similar lesion).

Local anæsthesia is used intradermally at the intended point of puncture and interstitially as required to render the procedure painless, at the discretion of the operator. A small puncture is made with a scalpel (since otherwise a disk of skin may be punched out by the needle and block it). The needle is introduced through the skin into the tumour and then the piston is withdrawn, producing suction by the syringe. Keeping the piston withdrawn in this way by means of the forefinger of the right hand braced against the end of the barrel of the syringe, the needle is pushed through the tumour and then withdrawn 1 or 2 mm. before pushing it again in a different direction. This manœuvre serves to cut off the thread of tissue in the syringe. Without withdrawing the needle from the tumour the process can be repeated two or three times. Then the suction is partly released and the needle withdrawn from the tumour. Complete release of suction may result in the tissue being left in the tumour. When the needle is being withdrawn from the skin the tissue may remain in the needle or be sucked into the syringe, which is filled with air and used to expel the tissue into a suitable tube. If the tissue is sucked into the syringe the pieces are best picked out with the scalpel and put into the specimen tube. If the pieces are very broken up, however, the syringe is best rinsed out vigorously with the formalin solution which is used as a fixative. The formalin is put into the specimen tube, which is then ready to go to the laboratory.

Drill Biopsy.—The technique is the same except that a squared-off needle of the type shown in *Fig.* 339 is used, the free end being bevelled and sharpened. This is introduced, using a pointed trocar, till it reaches the tumour. Then the trocar is removed and the end of the needle fixed in the chuck of a dental drilling machine. This is made to turn by an assistant or by the operator using the treadle of the machine. While it is rotating the needle is pushed steadily in a straight line into the tumour. When the limit of the tumour is reached the needle is withdrawn about 2 mm. and then, still rotating, pushed forward again in a different direction so as to cut off the thread of tissue. A syringe is then attached to the needle and suction applied during the withdrawal of the needle. The

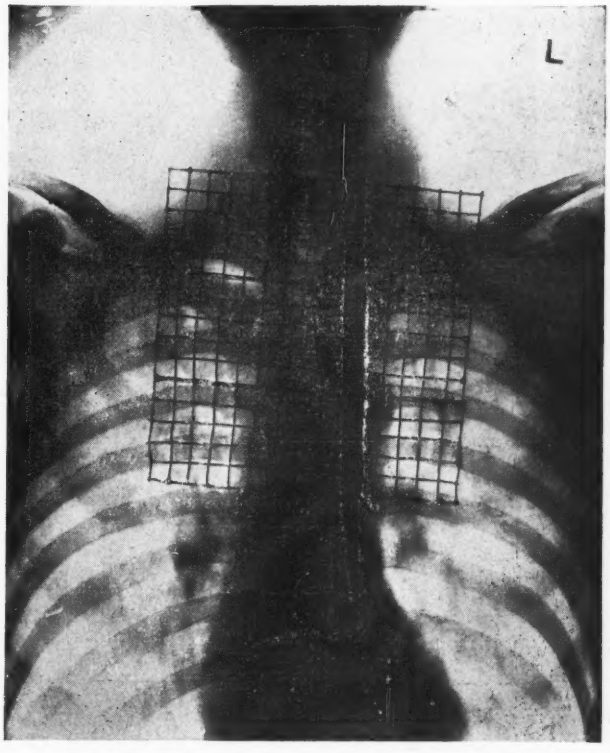


FIG. 342.—Wire grid used for localizing a spinal lesion. It is fixed to the skin of the back with strapping, therefore there is little distortion due to divergence of the X-ray beam.

thread of tissue may remain in the needle, but more frequently is sucked into the syringe. Its transfer to the specimen tube is effected as for aspiration biopsy.

Histological Technique.—This may be aimed at staining a fresh specimen or at providing a paraffin block. These methods are described elsewhere (Hermitte and Ellis*). The paraffin block method has been followed almost entirely in this series. Three methods have been used. Dr. Hermitte receives the specimen in a centrifuge tube and after centrifuging carries through the dehydration and embedding of the tissue in the tube, the block forming a cast of the centrifuge tube with the specimen at the top. Centrifuging is carried out at all stages of dehydration and the first two paraffins. Dr. Barrie receives the specimen in a flat-bottomed tube, in which it is allowed to settle. Dehydration and embedding are carried out in this tube, the block forming a cast with the specimen in the surface corresponding to the bottom of the tube. These pathologists have dealt only with aspiration biopsies. The technique used by Dr. Woods at the London Hospital has consisted in receiving the specimen in a

* Section in *Recent Advances in Pathology*. (In the press.)

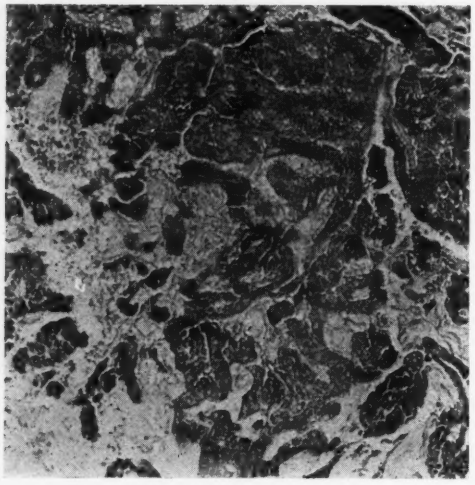


FIG. 343.—Adeno-basal-cell epithelioma below right orbit.

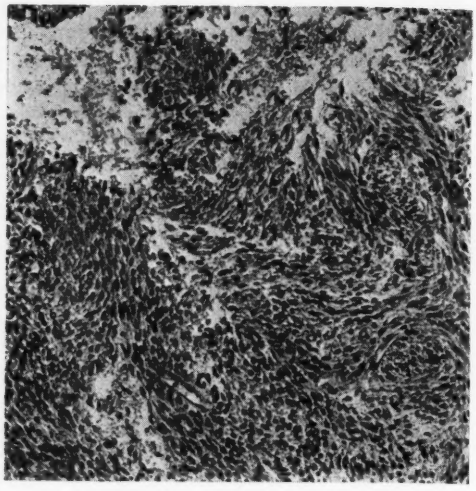


FIG. 344.—Fibrosarcoma.

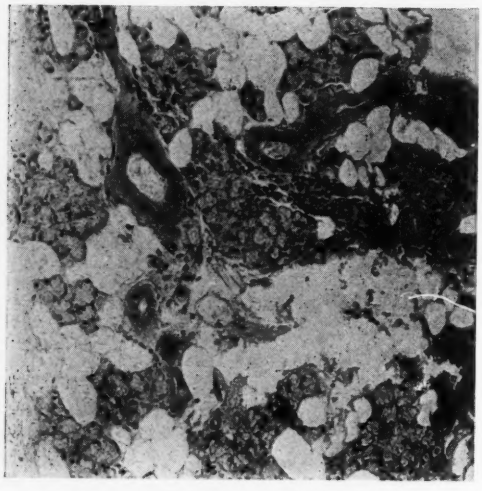


FIG. 345.—Normal parotid salivary gland.

FIG. 346.—Scirrhous adenocarcinoma of breast.

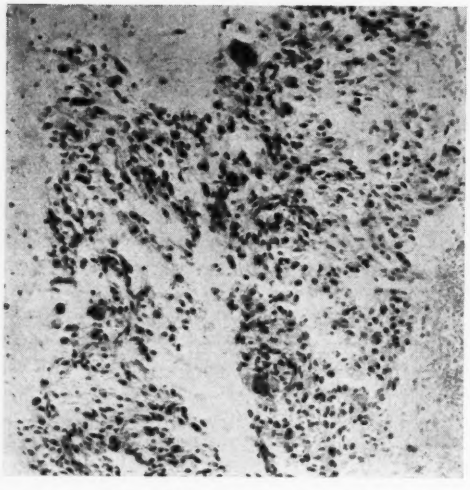


FIG. 347.—Pleomorphic fibrosarcoma aspirated from biceps swelling.

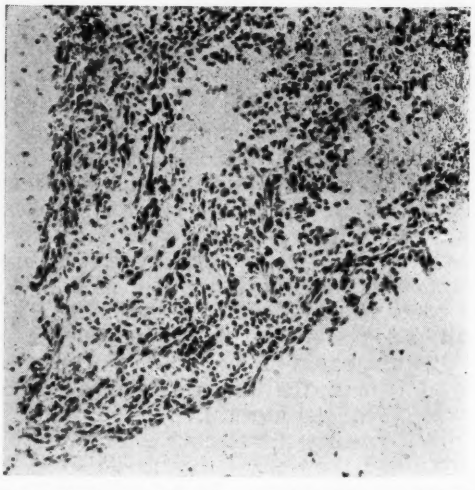


FIG. 348.—Granulation tissue aspirated from subacute inflammatory lesion of floor of mouth. (× 104.)

FIGS. 343–348.—ASPIRATION BIOPSIES.

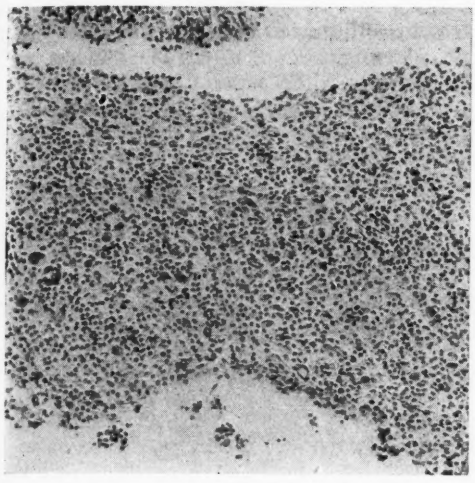


FIG. 349.—Portion of aspirated cervical lymph-node, showing typical changes of Hodgkin's disease, including several Dorothy Reede cells. (× 104.)

FIG. 350.—Portion of neurinoma of spinal nerve aspirated from swelling near angle of jaw, and showing typical palisading of nuclei of Schwann cells. (× 104.)

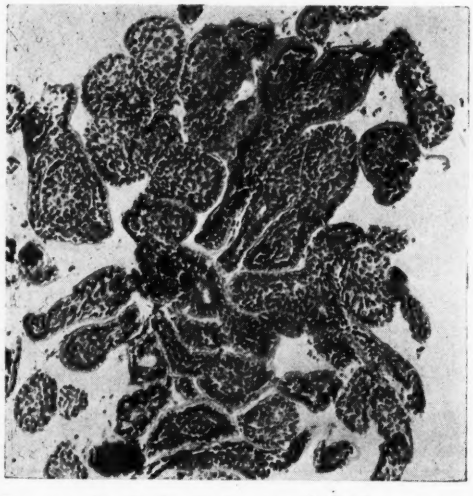


FIG. 351.—Basal-cell cylindroma aspirated from growth of temple. (× 104.)

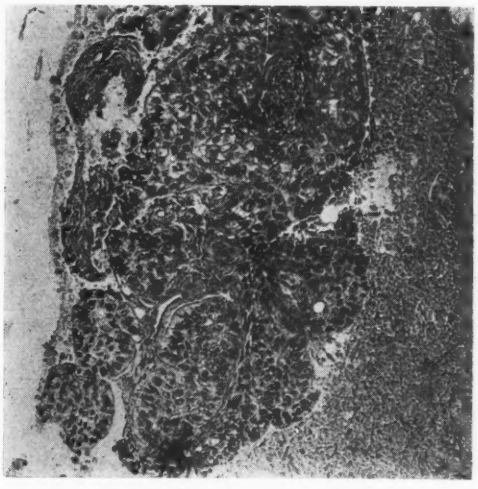


FIG. 352.—Grade 3 squamous epithelioma. (× 104.)

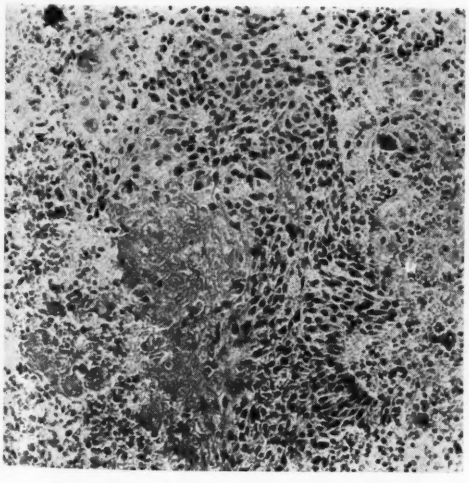


FIG. 353.—Grade 2 squamous epithelioma. (× 104.)

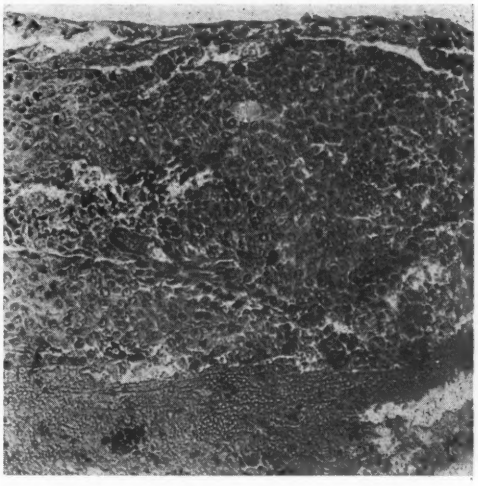


FIG. 354.—Grade 3 squamous epithelioma. (× 104.)

FIGS. 349–354.—ASPIRATION BIOPSIES.

small jar with a screw top. The pieces of tissue are picked out from it, dehydrated, and embedded in the usual way. The specimens thus dealt with have for the most part been from drill biopsies, which may be dealt with more easily; occasionally, however, centrifuging of the fixative has been required to obtain the specimen. The block having been obtained, it is cut in the usual way and sections stained as decided by the pathologist.

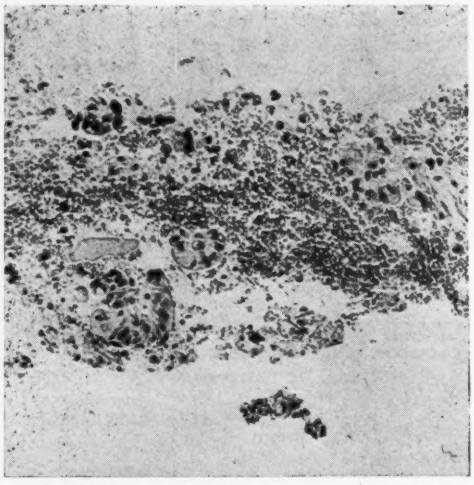


FIG. 355.—Caseous tissue (top) and tuberculous follicles with giant-cell system (bottom). T.B. (× 104.)

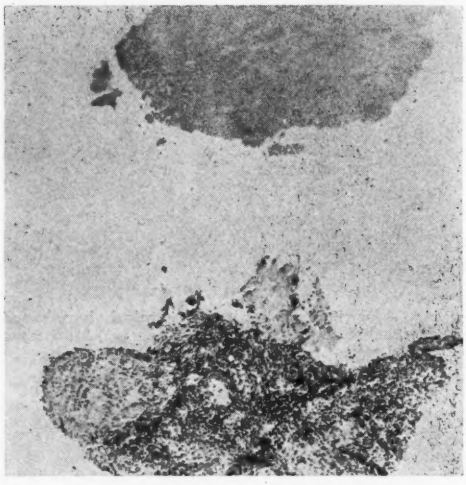


FIG. 356.—Adenocarcinoma invading abdominal operation scar. (× 104.)

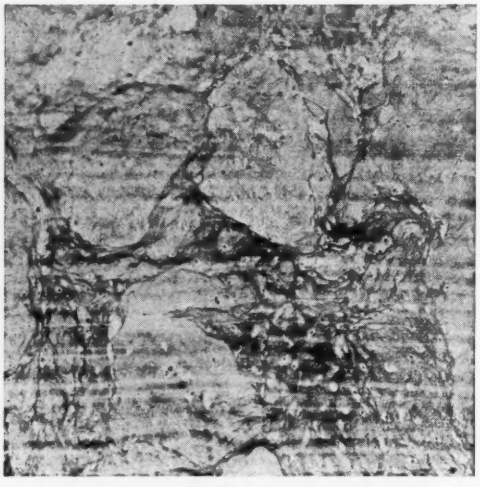


FIG. 357.—Aspiration from myxochondroma of head of humerus. (× 104.)

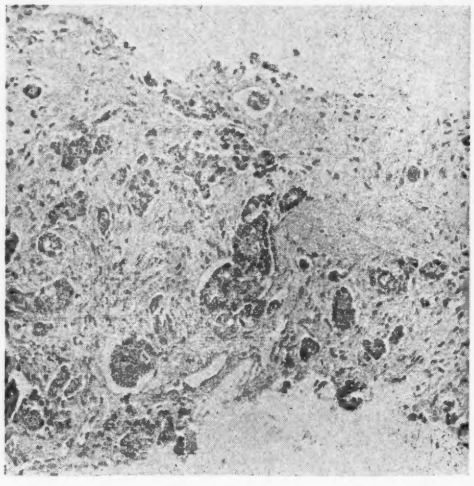


FIG. 358.—Metastases of breast carcinoma aspirated from sternum. (× 104.)

FIGS. 355–358.—ASPIRATION BIOPSIES.

Complications of the Clinical Technique.—These are of two types—those concerning the welfare of the specimen and those concerning the welfare of the patient.

The commonest difficulty is the presence of blood in the syringe. A relatively small amount of blood suffices to hide the specimen. The blood can easily be fixed, embedded, and cut, when a cellular specimen can easily be picked out under the microscope after staining. It is better, however, to pick out the specimen before sending it to the laboratory. To facilitate this it is wise to have about ½ c.c. of 3·8 per cent sodium citrate solution in the syringe to prevent clotting. If the citrated blood is then expelled on to a filter paper, the specimen of tissue can easily be picked out. Sometimes a swelling may prove unexpectedly to contain pus or fluid. A little pus or fluid can be put into a sterile tube for bacteriological examination and the rest treated as a histological specimen.

Complications affecting the welfare of the patient may be immediate or develop later.

Immediate complications are rare. Two cases only of this series suffered any immediate complication. One of these was a middle-aged woman with a lymph-node tumour in the left lower cervical region. In carrying out the aspiration biopsy with an excellent syringe, my forefinger slipped and the piston shot into the barrel of the syringe, driving the needle deep into the carotid artery and shattering the syringe. Blood spurted through the needle under

considerable pressure, but on withdrawing the needle and applying pressure at the site of puncture the bleeding rapidly ceased and did not even cause a hæmatoma. The second was a man of 52 with superior mediastinal obstruction and severe emphysema. After a painless and bloodless aspiration biopsy

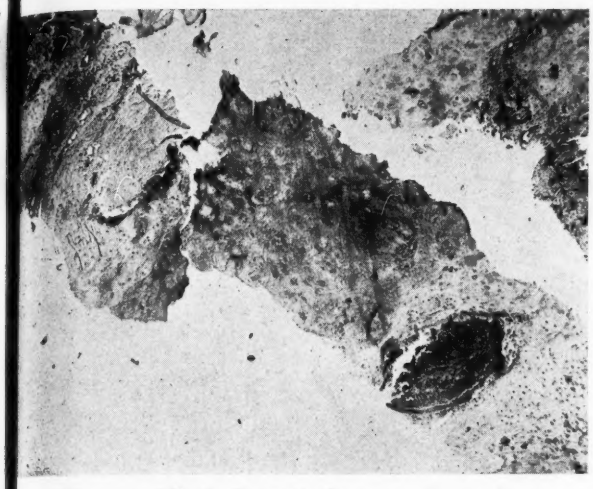


FIG. 359.—Mixed salivary-gland tumour. (× 97·5)

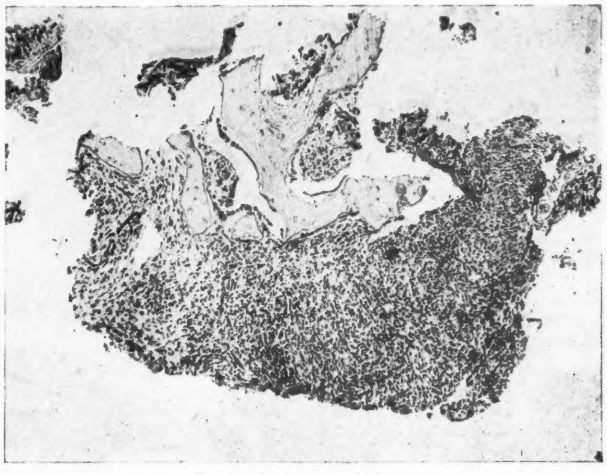


FIG. 360.—Tuberculous (?). (× 51.)

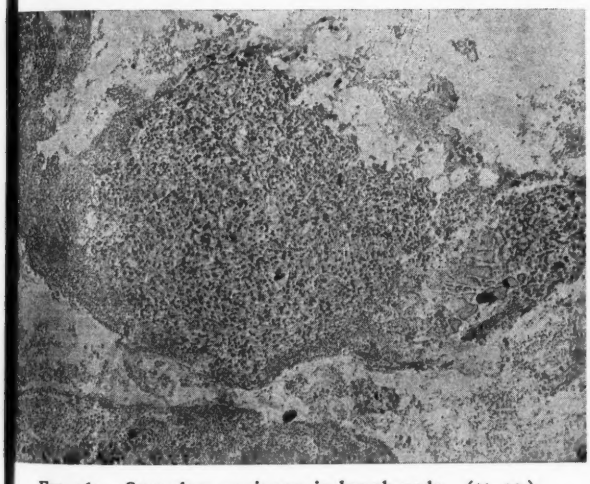


FIG. 361.—Secondary carcinoma in lymph-node. (× 33.)

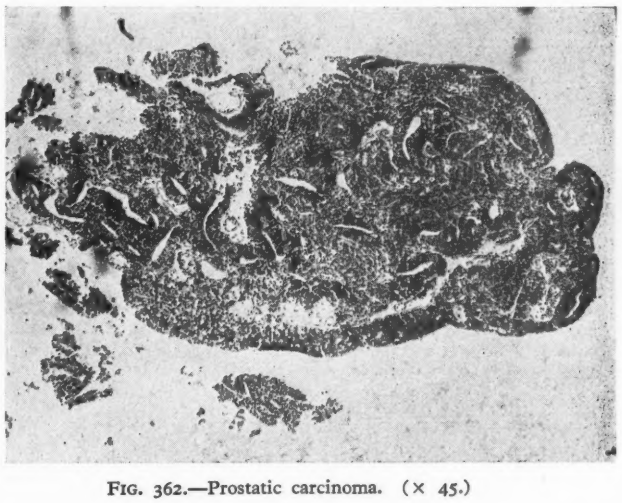


FIG. 362.—Prostatic carcinoma. (× 45.)

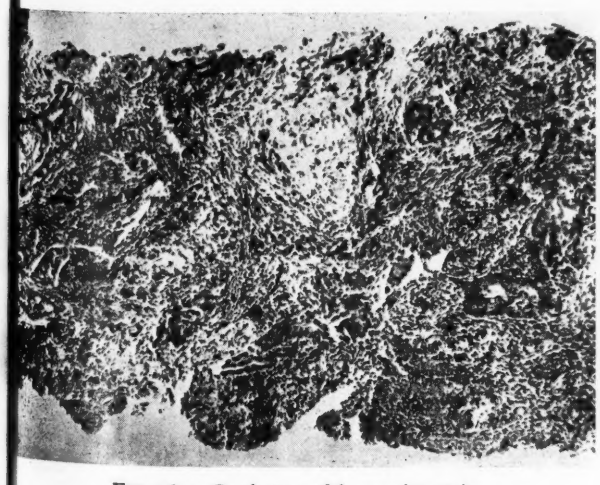


FIG. 363.—Carcinoma of lung. (× 57.)

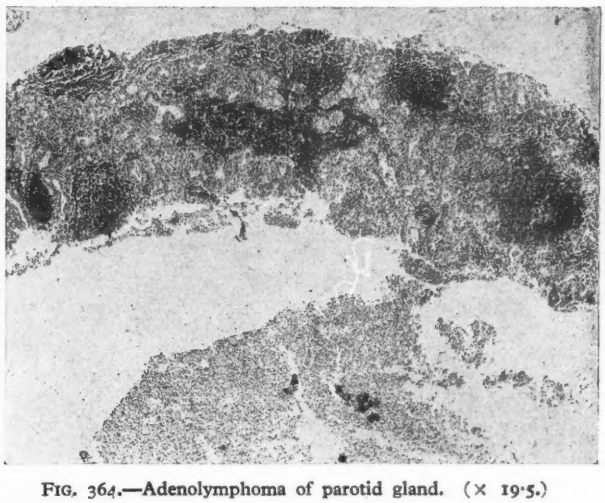


FIG. 364.—Adenolymphoma of parotid gland. (× 19·5.)

FIGS. 359–364.—DRILL BIOPSIES.

under local anæsthesia of a tumour of the left lung the patient rapidly became distressed in his breathing and died within fifteen minutes. The cause of death was collapse of the punctured lung due to rapid development of a pneumothorax, aggravating the superior mediastinal obstruction. This had occurred

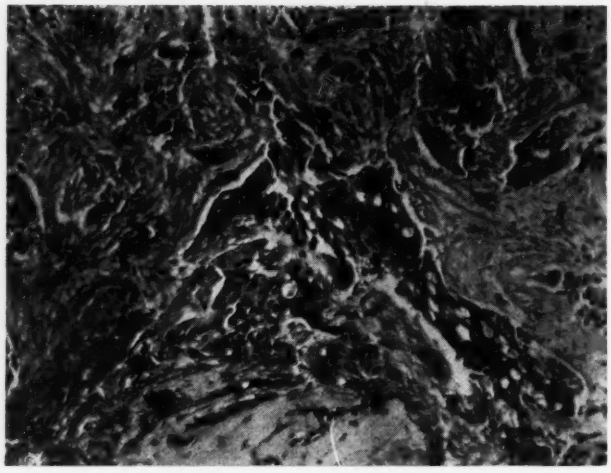


FIG. 365.—Scirrhous carcinoma of breast. (× 97·5)

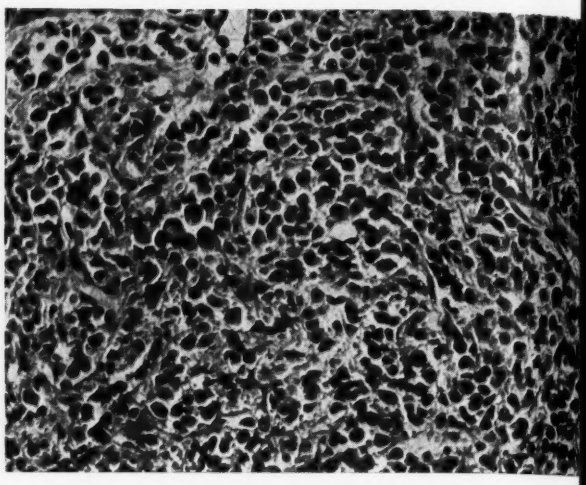


FIG. 366.—Myeloma. (× 240.)

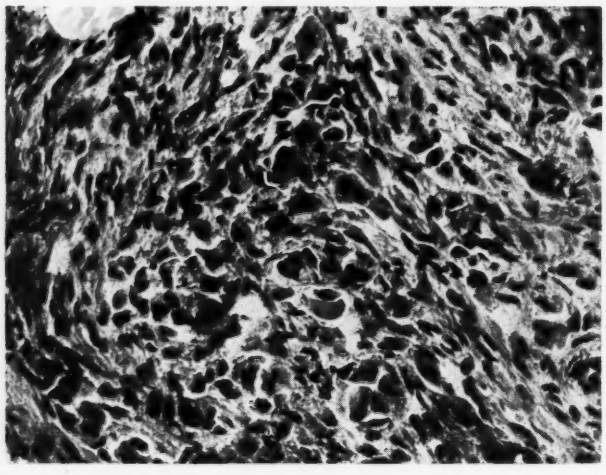


FIG. 367.—Carcinoma. (× 232·5.)

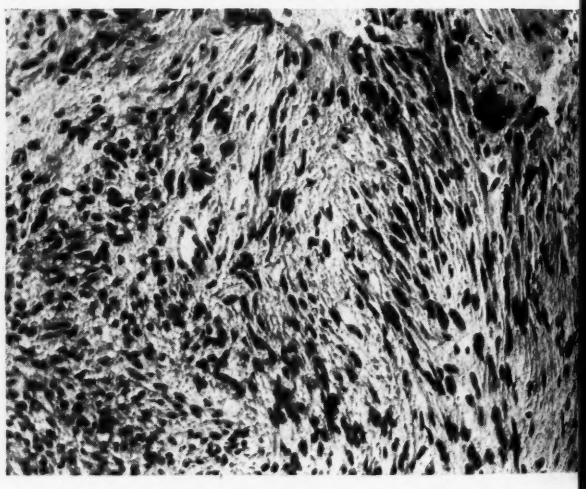


FIG. 368.—Sarcoma. (× 165.)

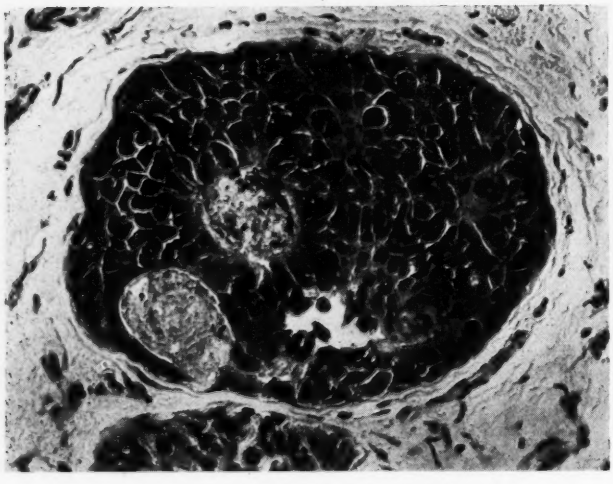


FIG. 369.—Breast: Solid polygonal-celled carcinoma. (× 262·5.)

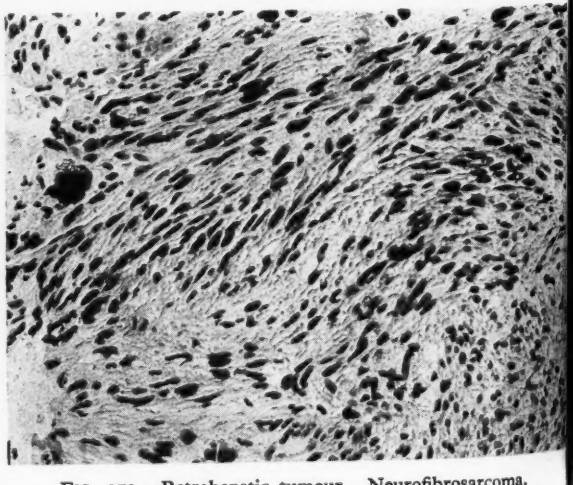


FIG. 370.—Retrohepatic tumour. Neurofibrosarcoma. (× 172·5)

FIGS. 365–370.—DRILL BIOPSIES.

because the puncture of the lung had rapidly developed into a tear owing to its emphysematous condition. If I had realized the cause in time, death might have been averted.

Late complications experienced comprise two only. One of these concerns a woman with carcinoma of the breast, one of the very few of these biopsies not carried out by me. During the two

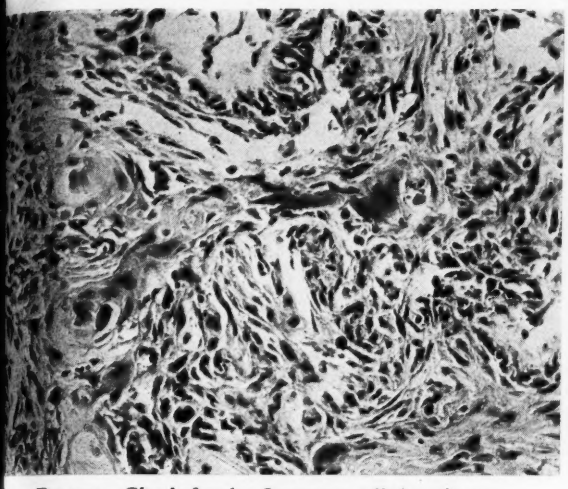


FIG. 371.—Gland of neck: Squamous-celled carcinoma. (× 150.)

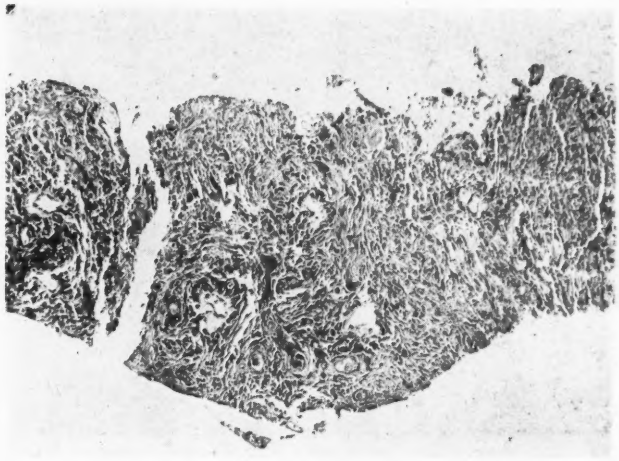


FIG. 372.—Gland of neck: Squamous-celled carcinoma. (× 49·5.)

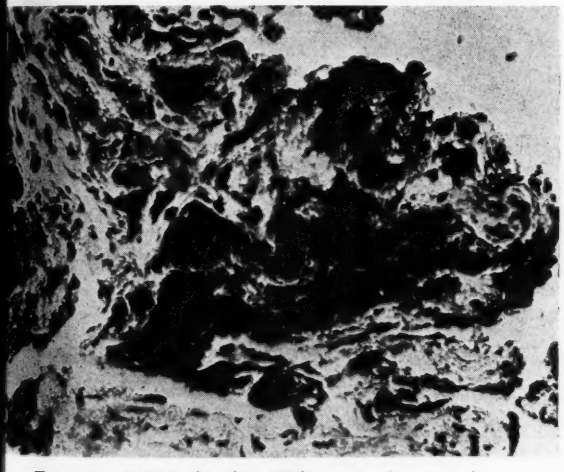


FIG. 373.—Gland of groin: Melanoma. (× 157·5.)

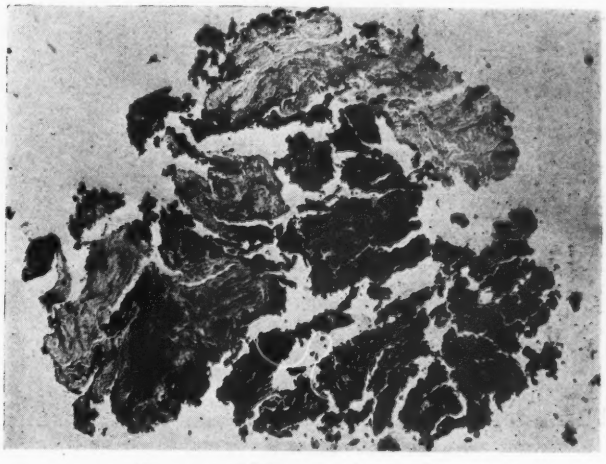


FIG. 374.—Melanoma. (× 55·5.)

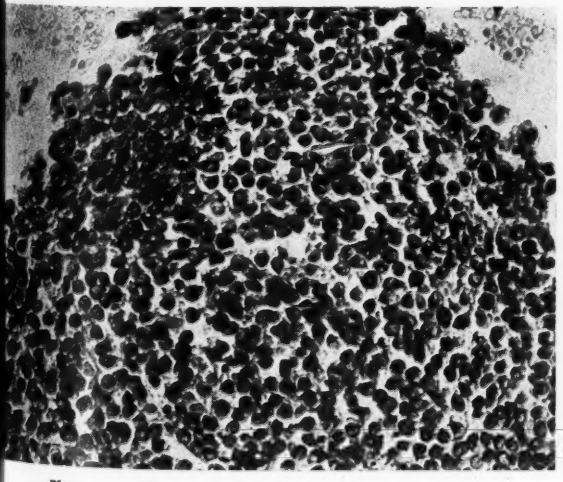


FIG. 375.—Bone: ? Ewing's tumour. (× 232·5)

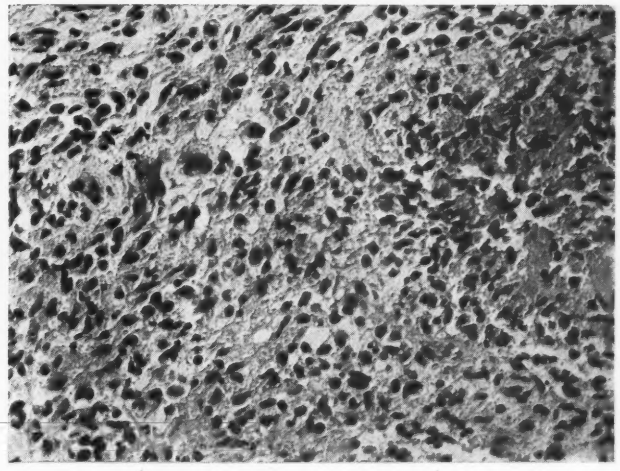


FIG. 376.—Gland of neck: ? tuberculosis. (× 225.)

FIGS. 371–376.—DRILL BIOPSIES.

weeks following the puncture the patient developed an acute infection spreading from the site of the puncture, the skin sloughing over a large area. The culture from this showed the infection to be diphtheritic, the operator in this case being a previously unsuspected carrier. The patient made a perfect

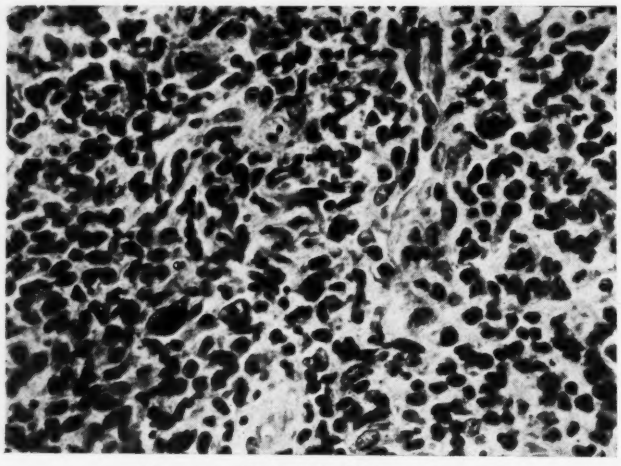


FIG. 377.—Hodgkin's disease. (× 337·5.)

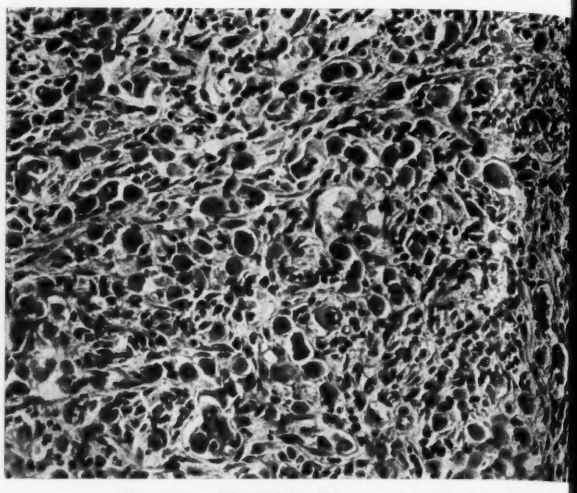


FIG. 378.—Hodgkin's disease. (× 169.)

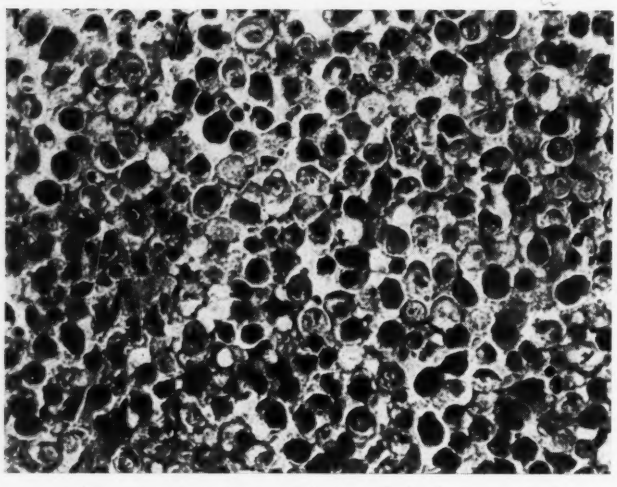


FIG. 379.—Secondary carcinoma of gland. (× 262·5.)

FIG. 380.—Parotid. (× 180.)

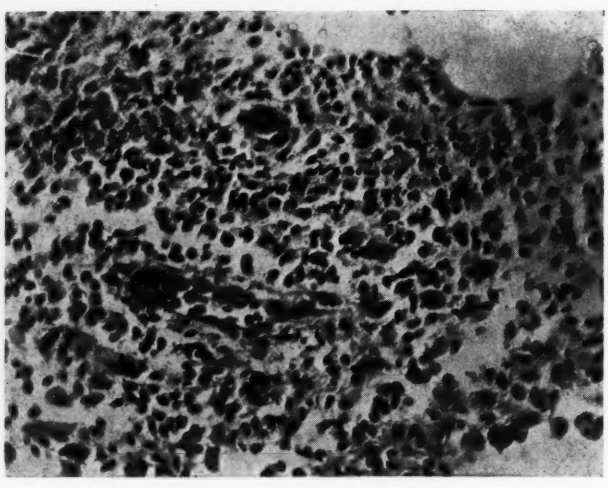


FIG. 381.—Femur: Pyogenic granulation tissue. (× 247·5.)

FIG. 382.—Femur: Pyogenic granulation tissue. (× 50.)

FIGS. 377–382.—DRILL BIOPSIES.

recovery. The other concerns a man of about 65 with a shadow of long duration in the mid-zone of the left lung. I was asked by the physician in charge to carry out an aspiration biopsy. This was done under evipan anæsthesia with the patient in his bed. The tumour was encountered, as usual, at the first

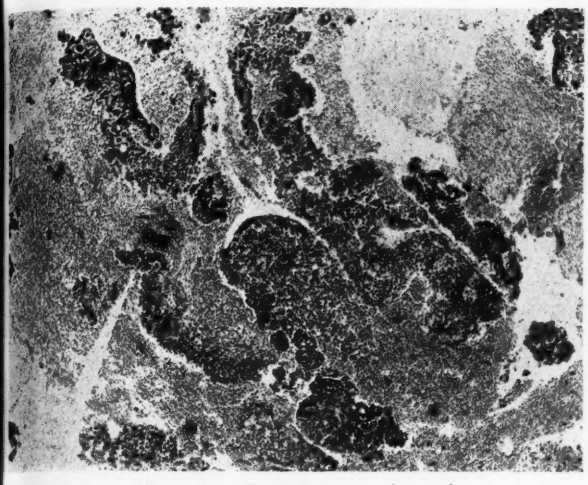


FIG. 383.—Osteoclastoma. (× 33.)

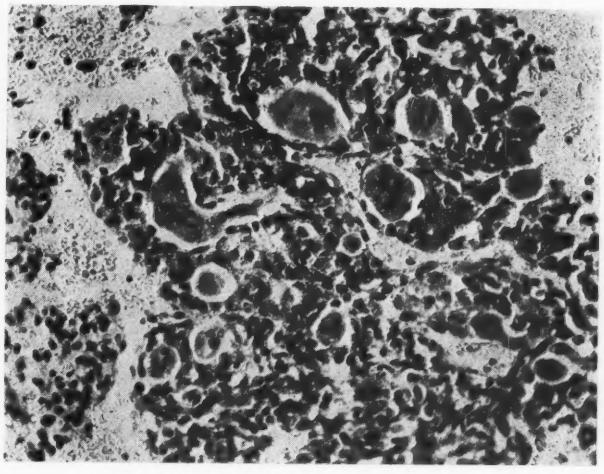


FIG. 384.—Osteoclastoma: ? malignant.

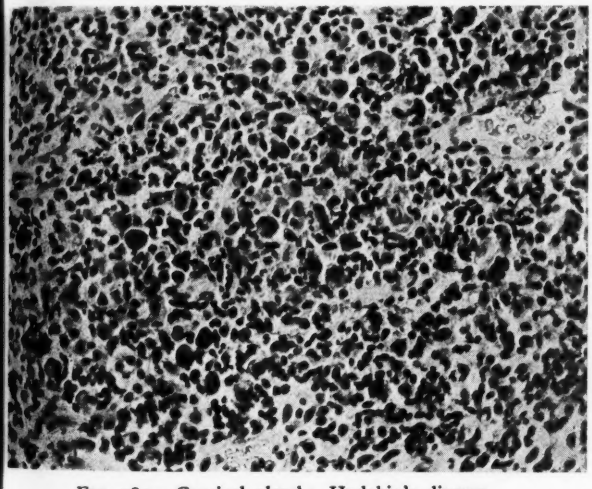


FIG. 385.—Cervical gland: Hodgkin's disease. (× 172·5)

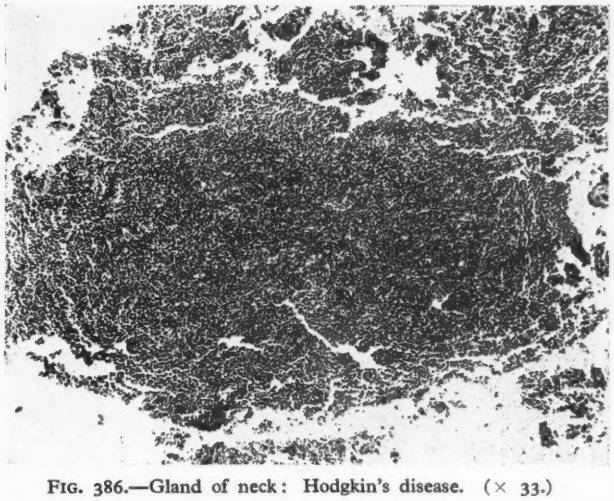


FIG. 386.—Gland of neck: Hodgkin's disease. (× 33.)

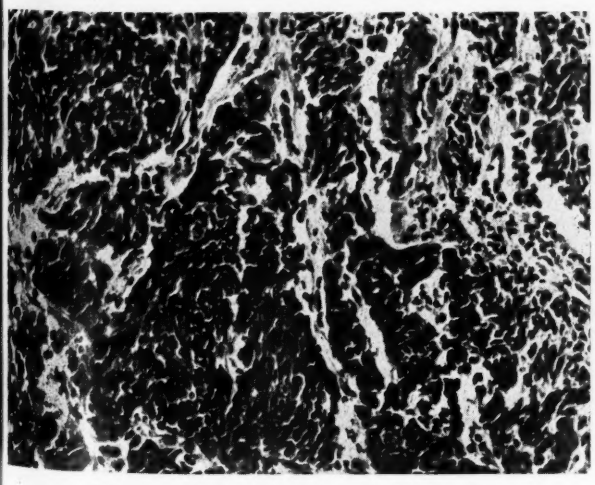


FIG. 387.—Liver: Secondary carcinoma of lung. (× 210.)

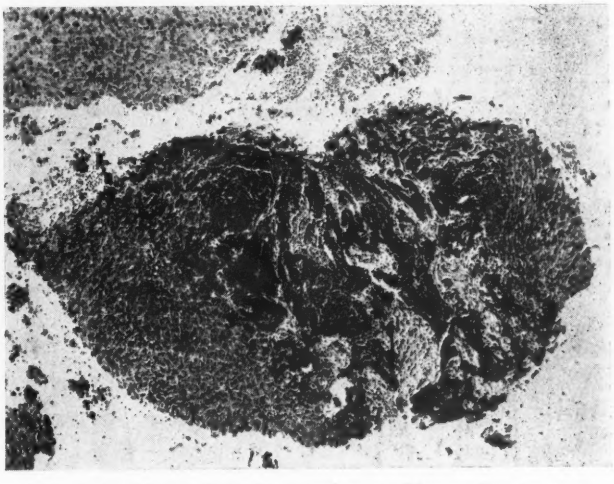


FIG. 388.—Liver: Secondary carcinoma of lung. Low-power view of *Fig.* 387. (× 42.)

FIGS. 383–388.—DRILL BIOPSIES.

puncture and thick yellow pus sucked into the syringe. Nothing further was done, but the patient developed signs two or three days later of a pneumonitis from which he died within two weeks.

No other complications have occurred.

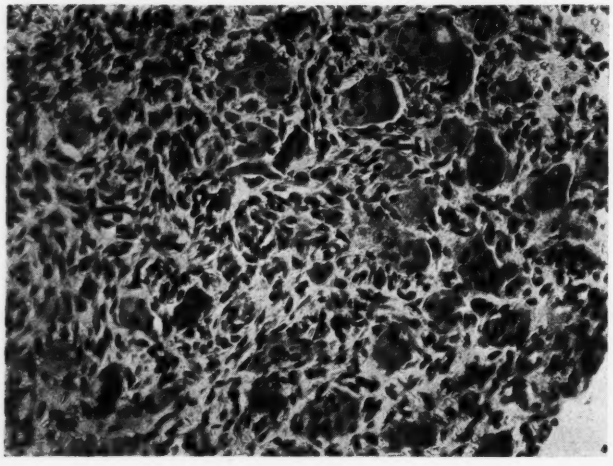


FIG. 389.—Osteoclastoma. (× 157·5)

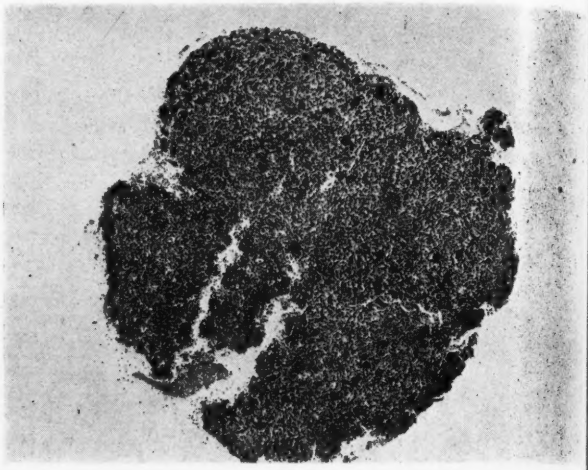


FIG. 390.—Osteoclastoma. (× 30.)

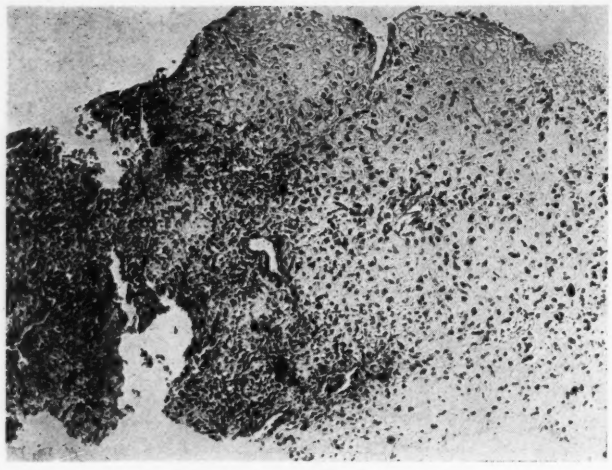


FIG. 391.—Sarcoma. (× 45.)

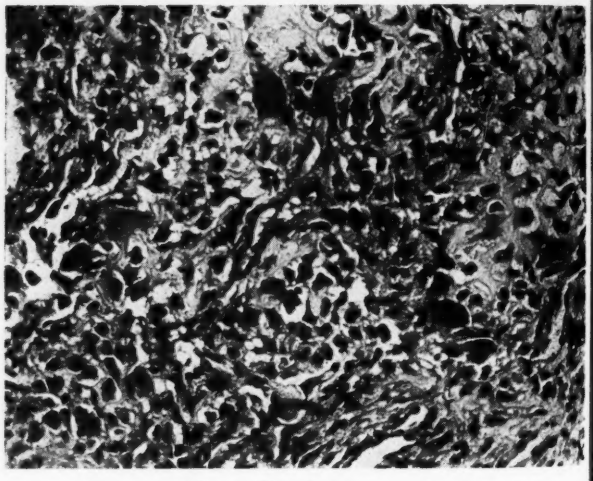


FIG. 392.—Tibia: Chondrosarcoma. (× 180.)

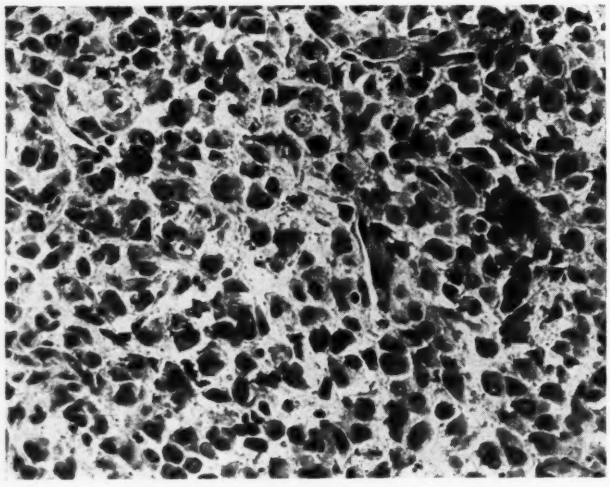


FIG. 393.—Thyroid: Polygonal-celled carcinoma. (× 217·5.)

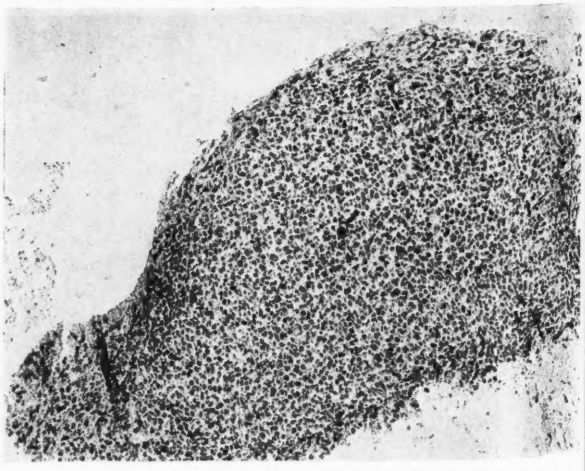


FIG. 394.—Carcinoma of thyroid. (× 45.)

FIGS. 389–394.—DRILL BIOPSIES.

Technical Advantages of Drill Biopsy over Aspiration Biopsy.—The advantages of drill biopsy in my experience are :—

1. The operator has greater control, not having to apply suction at the time of introducing the needle. This is a great psychological advantage, giving more confidence to the operator, and thus making the procedure more widely applicable.

2. A larger piece of tissue is obtained which, besides enabling the pathologist to give a greater number of positive reports, seems to permit also a greater degree of confidence in individual reports.

Both procedures may be carried out equally quickly, although the drill biopsy requires more specialized apparatus.

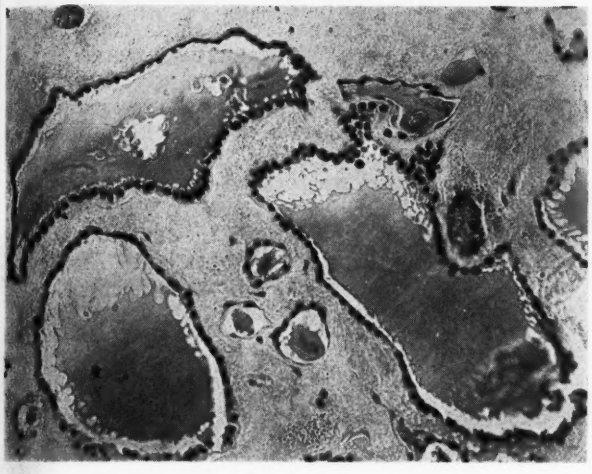


FIG. 395.—Thyroid gland. (× 150.)

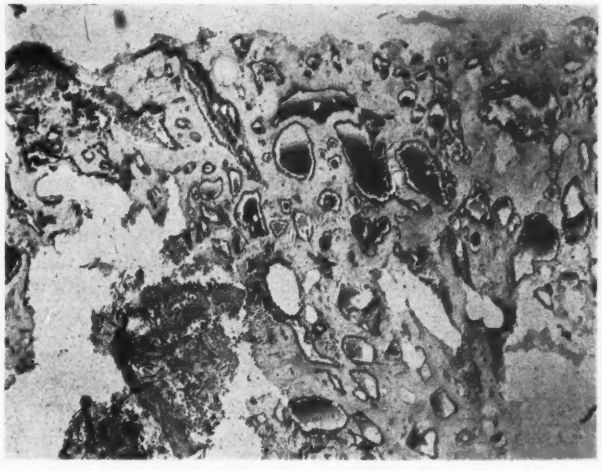


FIG. 396.—Thyroid gland: Low-power view of *Fig.* 395. (× 36.)

FIGS. 395, 396.—DRILL BIOPSIES.

By using a motor to drive the drill it seems likely that the ease of operation might be increased, the speed of rotation of the drill controlled, and possibly an optimum speed decided upon.*

In conclusion, I should like to thank all those who have helped to make this account possible, especially Dr. Hermitte, Dr. Barrie, and Dr. Woods, Pathologists at the Sheffield Royal Infirmary, the Sheffield Royal Hospital, and the London Hospital respectively, without whose patience and skill the clinical work would have been useless. Dr. Hermitte, who provided the photographs of histological sections of aspiration biopsies, Mr. Trent, physicist at the London Hospital, and Mr. McOwan have been responsible for producing the drilling needles which I have used, while Mr. John King has been responsible for the photographs of the drill biopsies. Miss Wolff helped by finding the fields to be photographed. Dr. Walter Shanks, my assistant at the London Hospital, helped with the analysis of the aspiration biopsies. All the members of the staff of a department help in one way or another to make possible any publication, and of others I would like to thank especially the Sister, Miss D. Hodgson, and my secretary, Miss Hallett.

* Since this article went to press a motor has been used to rotate the drill for months. It is a distinct improvement in technique.

REFERENCES

BINKLEY, J. S. (1939), *Amer. J. Cancer*, **36**, 193.
CHRISTIANSEN, H. (1940), *Acta radiol., Stockh.*, **21**, 349.
— — (1942), *Ibid.*, **23**, 147.
COLEY, B. L., SHARP, G. S., ELLIS, E. B. (1931), *Amer. J. Surg.*, **13**, 215.
FERGUSON, R. S. (1930), *Ibid.*, **9**, 507.
HAAGENSEN, C. C., and STOUT, A. D. (1942), *Ann. Surg.*, **116**, 801.
IVERSON, P., and ROHOLM, K. (1939), *Acta med. scand.*, **102**, 1.
KHANOLKAR, V. R., and SHARPURE, V. V. (1944), *Indian Physician*, **3**, 6.
MARTIN, H., and ELLIS, E. B. (1930), *Ibid.* **13**, 169.
— — — — (1934), *Surg. Gynec. Obstet.*, **59**, 578.
— — — — (1940), *Treatment of Cancer and Allied Diseases*, **1**, 72.
— — and STEWART, F. W. (1936), *Amer. J. Roentgenol.*, **35**, 245.
MARZLOFF, K. H. (1923), *Bull. Johns Hopk. Hosp.*, **34**, 141.
OTTOLENGHI (1943), *Radiologia*, **6**, 288.
PATERSON, R., and NUTTALL, J. (1939), *Amer. J. Cancer*, **37**, 64.
SAYAGO, C. (1942), *Amer. J. Roentgenol.*, **48**, 78.
STEWART, F. W. (1933), *Amer. J. Path.*, **9**, 801.

RENAL ANOXIA AND THE TRAUMATIC URÆMIA SYNDROME

By Squadron Leader E. M. DARMADY, R.A.F.V.R.

PATHOLOGIST TO ROYAL AIR FORCE HOSPITAL, WROUGHTON, AND ROYAL AIR FORCE CASUALTY CLEARING STATION IN ENGLAND

It was the experience of the staff at the R.A.F. Casualty Clearing Station in England that successful surgery was often marred by the onset of uræmia which sometimes proved fatal. The Unit was dealing with casualties evacuated by air from Western Europe and only those cases which were unfit to travel further or which required immediate surgery were admitted to hospital. Among 10,000 casualties admitted there were 44 deaths, and in 12 of these cases death was directly due to renal failure.

As it was felt that an early report might assist in the recognition of similar cases, full details were published of the first 6 of these fatal cases, and of 2 cases in which renal failure was recognized clinically, and which recovered (Darmady et al., 1944).

Biochemical investigations were continued and the present report deals with a further 6 cases of fatal traumatic uræmia and 3 more cases of uræmia with recovery. It is clear that the biochemical course of all the cases and the histological changes found post mortem in the fatal cases are sufficiently similar to warrant the assumption that some common factor was operative in producing the uræmia.

DESCRIPTION OF THE SYNDROME

The clinical features are characteristic and in the series under consideration all were severely wounded men who had suffered considerable loss of blood, accompanied by shock requiring energetic resuscitative measures. Thus in many cases prolonged periods of hypotension were recorded. Major vessels were involved in the injury in 9 cases and 14 had fracture of long bones. Except for *Case* 17, no crushing injury or undue damage to muscle was observed. Dehydration was particularly noticeable in 3 cases. Blood transfusion had not been given in *Case* 15; the remainder had received Group 'O' blood supplied by the Army Blood Transfusion Service. Transfusion reactions were observed in 3 cases only. Sulphanilamides had been given in 10 cases.

The onset of symptoms took place between the second and sixth day after injury. The first sign was usually anorexia, with a tendency to hiccup, and this was followed in the course of a few days by copious vomiting. The tongue was dry and brown, and the patient was drowsy, with attacks of hallucinations. Cyanosis was frequently present. Occasionally a petechial rash was seen over the upper parts of the body. This was thought to be a manifestation of fat embolism (a common feature in severely wounded casualties). During the prodromal period oliguria was present, although this was not always obvious unless a fluid intake and urinary output chart was kept. Anuria developed in *Case* 14. Anæmia was also present, and once uræmia was apparent the blood-pressure was frequently raised (7 cases). The condition of the patient rapidly deteriorated, and within ten days from the injury or exciting cause death occurred. In those cases that recovered the sudden improvement was dramatic and occurred about the sixth to the eighth day.

Full details of these cases have been recorded elsewhere (Darmady, 1946).

BIOCHEMICAL CHANGES

All the cases recorded had an enormous rise in blood-urea, oliguria, and a disturbance of electrolyte balance.

Blood-urea.—Estimation of the blood-urea is of considerable importance, since it is this which differentiates the case of traumatic uræmia from other severely wounded men. *Fig.* 397 shows the blood-urea estimations made on 79 seriously wounded casualties. These cases were selected either because they were on the dangerously ill list or had ischæmic limbs. The estimations were carried out on admission to hospital, which was between 2 and 5 days after wounding, at which time traumatic uræmia, if present, would be already apparent. *Fig.* 397

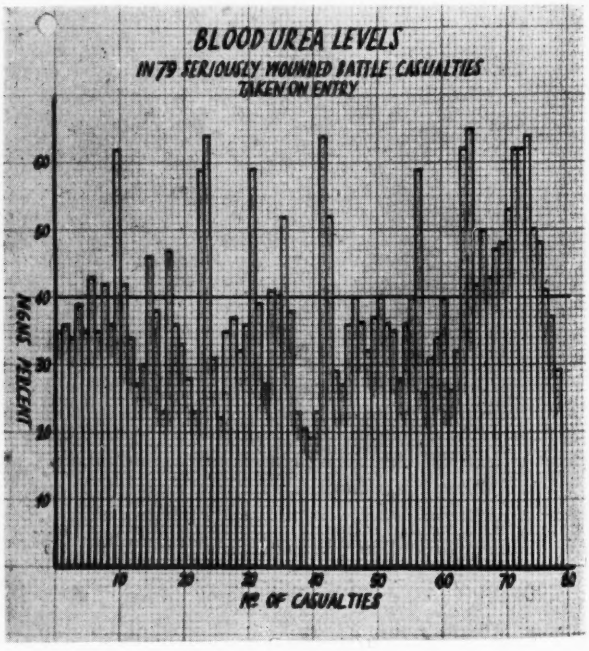


Fig. 397.—Blood-urea levels, taken on entry, in 79 seriously wounded battle casualties.

shows that 28 (35 per cent) had a raised blood-urea, and approximately 60 per cent of the total number examined had a diminution of urea clearance. The highest blood-urea recorded from this series was only 66 mg. per cent. In *Table I* it will be seen that the initial values in the 11 cases of fatal traumatic uræmia are on a considerably higher level, and that there is a progressive rise of the blood-urea. This high level may be maintained for several days before death occurs. *Table II* shows the blood-urea from the cases which recovered. In this series the

blood-urea values tend to be lower, and rise to a peak about the sixth to the eighth day, when there is a sudden fall. This fall is sometimes dramatic (*Case* 16) and suggests that a 'crisis' has occurred. The significance of this will be discussed later.

Oliguria.—This was present in all cases and had some curious aspects, since the volume of urine passed bore a relationship to the intake of fluid. This is shown in *Table III*, where the fluid intake and output is recorded during the first three days that *Case* 10 was under observation. The first figure in the intake column refers to the volume of isotonic saline given intravenously, and the second is the amount taken by mouth. In the output column the first figure is the amount of urine passed; the second the quantity of vomit or of fluid removed by gastric suction. These figures demonstrate clearly the retention of fluid within the body and the relationship of urine secreted to fluid intake. This table also shows other characteristic features of traumatic uræmia. The urinary urea remains constant at values at between 1·8 and 2·1 mg. per cent in spite of the fact that the blood-urea value swings between 496, 461, and 473 mg. per cent. The specific gravity of the urine remained between 1014 and 1018. The *p*H of the urine did not vary with the alteration in alkali reserve (44–66 vols. per cent) but remained at 6·4–6·6–6·8.

Table I.—Blood-urea Findings in Fatal Cases of Traumatic Uræmia

Case No.	Nature of Injury	Days after Wounding													
		4	5	6	7	8	9	10	11	12	13	14	15	16	Later
1	Compound fracture, femur; gunshot wound, knee-joint	72	Died												
2	Left forearm and buttock; traumatic amputation of left leg		240	285	Died										
3	Gunshot wound, foot; compound fracture of radius and ulna	68	270	408	Died										
4	Thigh; gas gangrene; *Cl. septique*					138	260	424	Died						
5	Compound fracture, tibia and fibula					490	480	600	730	960	1100	720	774	Died	
6	Chest, buttock, and loin; compound fracture of humerus and radius and ulna				103	60	91	148	219	Died					
9	Compound fracture of femur with multiple flesh wounds	91	163	181	156	309		Died							
10	Popliteal fossa; amputation of leg					496	461	473	451	380	336	280	488	520	Died
14	Multiple gunshot wound of abdomen; duodenal fistula			172	296		231	351	Died						
15	Multiple burns		151		Died										
17	Extensive crush injury of leg	85	140	228	263	272	344	296	Died 452						

The progressive rise of blood-urea is seen in 11 fatal cases of traumatic uræmia. This is striking, not only because of the very high levels which may be attained, but because of the ability of the body to survive these levels for several days. The variety and extent of the wounds are noticeable.

Table II.—Blood-urea Findings in Cases of Traumatic Uræmia with Recovery

Case No.	Nature of Injuries	Days after Injury																						
		4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
7	Compound fracture of femur involving vessels				102		148					86				32								
8	Gunshot wound, leg, neck, chest wall, forearm, and fracture of big toe								86		59													27
11	Compound fracture of leg, traumatic amputation of right leg; wounds of thigh and buttock			158	120	112	109	98	61	46														
12	Fracture of femur; multiple wounds of buttocks			91	135	106	95	104	92	72		50		38										
16	Left buttock; left thigh	500	480	820	220	160	124				93					73		50				40		

The blood-urea values in the cases with recovery are on a lower level than those seen in *Table I*. The dramatic fall observed in *Case* 16 suggests that a 'crisis' had occurred.

Disturbance of Electrolyte Balance.—This presents several problems, since not only was there an acidosis but also hypochloræmia. It was easy to alter the alkali reserve by treatment with intravenous sodium lactate, but the restoration of the alkali reserve was difficult to maintain. Even if the electrolyte balance of the blood is corrected early, the disease is still progressive and there is a continued rise in blood-urea (*Cases* 9, 10, *Table I*).

Table III.—Biochemical Data from *Case* 10.

	Days after Wounding		
	11	12	13
Blood-urea (mg. per cent)	496	461	473
Alkali reserve (vol. per cent)	44	—	64
Intake (oz.)	100 80 + 20	100 20 + 80	90 70 + 20
Output (oz.)	72 58 + 14	64 60 + 4	66 50 + 16
No. of specimens of urine passed	7	5	9
Specific gravity	1018–1012	1018–1012	1018–1012
Urinary urea (mg. per cent)	2·1–1·8	1·9–1·8	1·9–1·8
*p*H	6·4	6·6–6·8	6·6–6·8

The biochemical data from *Case* 10 is shown on the eleventh, twelfth, and thirteenth days after wounding (*Table III*). The first figure in the intake column refers to the volume of isotonic saline given intravenously and the second is the amount taken by mouth. In the output column the first figure is the amount of urine passed; the second the quantity of fluid vomited or removed by gastric suction. The retention of fluid within the body is noticeable. Each specimen of urine passed was tested separately for specific gravity, *p*H, and urinary urea. The figures recorded represent the maximum variation each day.

Case 9.—Private K. was admitted, under Sq./Ldrs. Morel and Belcher, on the fourth day after receiving a perforating wound of his popliteal fossa. Amputation of the leg was done after resuscitation with three pints of blood and four pints of plasma. Post-operative shock was treated with a further two pints of blood. On the third day after wounding his blood-urea was 91 mg. per cent (*Fig.* 398). On the fourth day after wounding he was transferred to the R.A.F. Hospital. He was hiccuping, and it was found that his serum alkali reserve and his blood-chlorides were subnormal. 1000 c.c. of $\frac{1}{6}$-mol. sodium lactate were given, and there was an immediate and dangerous swing from acidosis to alkalosis. This was corrected with 1500 c.c. of isotonic saline daily given by mouth, but in spite of this there was a progressive rise of blood-urea, which reached 303 mg. per cent on the day before death.

Case 10.—C.S.M. E. showed the difficulty of maintaining the electrolyte balance. He was admitted on the tenth day after receiving a through-and-through wound of the popliteal fossa with a compound fracture of the femur. There had been severe and prolonged shock immediately after wounding and he had received extensive resuscitation. On admission he was cyanosed; his tongue was dry and very furred, and he had persistent hiccup. His blood-chloride and his alkali reserve were subnormal (*Fig.* 399). In this case alkalis were used with caution, and he was treated by isotonic saline intravenously; his daily intake being maintained between 90 and 100 fl. oz. The patient showed satisfactory progress, and the blood-urea fell considerably. On the sixteenth day after wounding he had a severe secondary hæmorrhage which necessitated an emergency amputation above the site of fracture. His general condition was such that he was given three pints of blood. Once more he developed acidosis and was therefore given 500 c.c. of $\frac{1}{6}$-mol. sodium lactate intravenously. His condition deteriorated; his blood-urea rose, and he died on the nineteenth day.

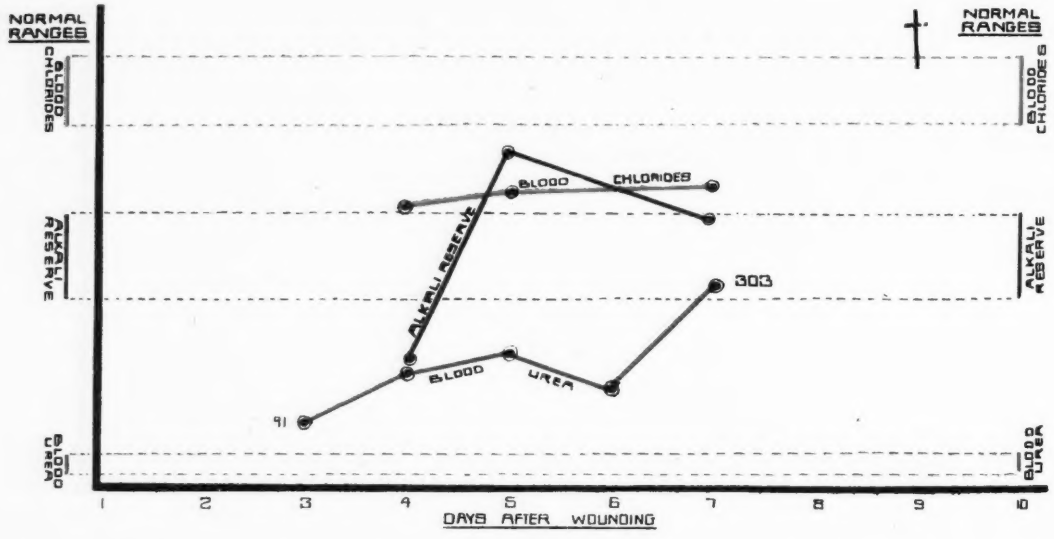


Fig 398.—Shows the biochemical course of *Case* 9. There is a progressive rise in the blood-urea. On the fourth day the blood-chlorides are low and the patient is in a state of acidosis. This case illustrates the ease with which the patient can be brought from acidosis to a state of alkalosis.

It was found in other cases that the *p*H of the urine failed to reflect the level of alkali reserve, and it was usual for patients to secrete acid urine after the alkali reserve had been increased or while in alkalosis; even cases of a milder nature with recovery showed this phenomenon.

Case 11.—Sapper B. was admitted under the care of Wing Commander J. C. Scott and Sq./Ldr. Vitek on the seventh day after wounding. He had received wounds of the left leg and buttocks in a mine explosion. Resuscitative measures had included 2 pints of blood and 3 pints of serum intravenously; débridement had been carried out and his leg placed in plaster. At the time of admission (seventh day after wounding) his condition was serious. The blood-urea was 120 mg. per cent (*Fig.* 400). The CO_2-combining power of the serum and blood-chlorides was low. He was secreting a urine of *p*H 5·2. He was given saline by mouth and 1 litre of ⅙-mol. sodium lactate solution and 2 litres of glucose-saline intravenously, and on the day following his alkali reserve had risen to 68 vol., the *p*H of his urine being 5·4. Treatment was continued on the same lines and the alkali reserve rose to 88 vol., the urine still remaining acid. An improvement in his general condition was noted. His blood-urea had returned to normal limits on the twelfth day, but it was not until the thirteenth day that the urine became alkaline. He progressed to complete recovery.

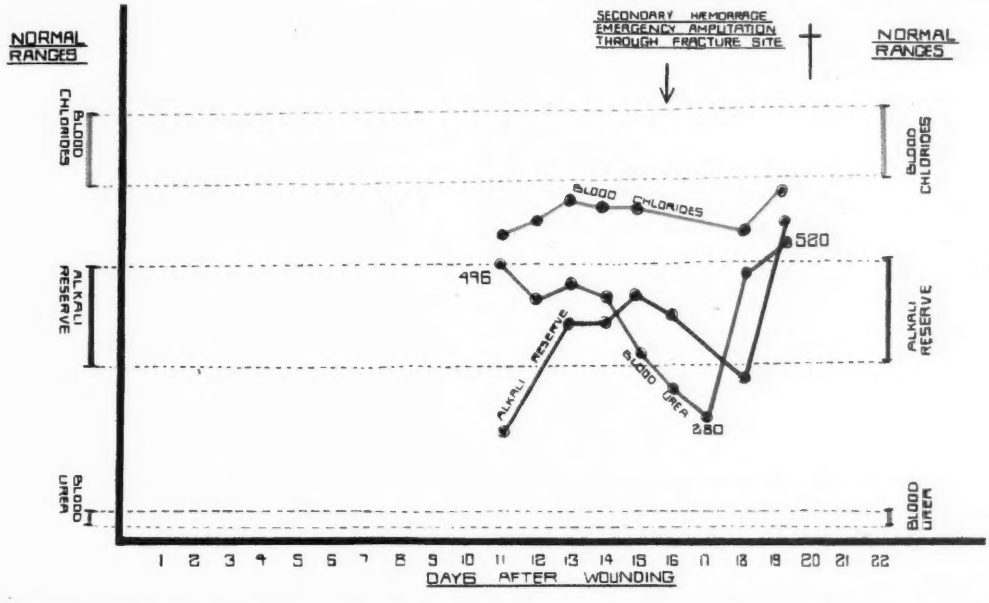


FIG. 399.—Biochemical course of *Case* 10. The first estimations on the eleventh day after wounding show a greatly increased blood-urea and a considerable fall in the blood-chlorides and CO_2-combining power. There was a temporary improvement, and following a secondary hæmorrhage the blood-urea rose. Death resulted from uræmia with alkalosis.

In none of the fatal cases did the blood-chloride value return to normal levels, whereas in the biochemical investigation of severely wounded battle casualties it was found that although initially the blood-chloride was low there was a progressive increase to within normal limits. Further, the use of alkalis was liable to cause a severe alkalosis. This would appear to be important, and will be commented upon in the discussion.

Further biochemical abnormalities were the rise in serum-potassium and inorganic phosphate; in some cases there was a fall in serum-sodium, and in the later cases serum-calcium, but the last four observations were infrequent and cannot be given in full.

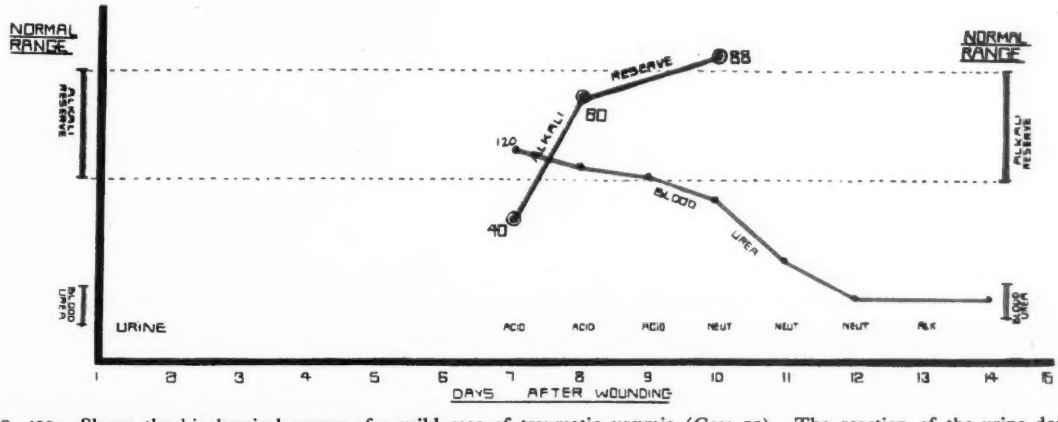


FIG. 400.—Shows the biochemical course of a mild case of traumatic uræmia (*Case* 11). The reaction of the urine does not reflect the changes in the level of the CO_2-combining power of the blood. The patient is in alkalosis on the tenth day and secretes a neutral urine. When the blood-urea has returned to normal the urine becomes alkaline.

AUTOPSY FINDINGS

Macroscopical.—The post-mortem examinations were carried out as soon as possible after death. In some cases amputation had been performed. The degree of muscle damage was slight, except in

Case 17. The tissues and muscle groups adjacent to the wounds were carefully incised in order to assess the amount of necrosis, which was insignificant, and to exclude the presence of concealed hæmatoma. Search was made for other internal injuries.

In *Cases* 5, 9, 10, 15, and 17 there had been evidence of ventricular failure in life, and this was confirmed post mortem. In the majority of cases minor degrees of fat embolism were found, particularly in the lungs.

The kidneys of all cases were similar. In those dying early, the capsule stripped easily, the cut edges were everted. The demarcation was accentuated by the yellow glistening texture of the cortex. In those cases dying later, the kidneys were cyanotic, although the yellow refractile surface of the cortex was again noticeable. The capsule was wrinkled and suggested that the kidney had been under tension. No other gross lesions were found in other organs.

Microscopical.—Microscopical examination of the liver, spleen, heart, brain, lungs, suprarenals, and kidneys were carried out in every case. The last four organs were also examined for fat. Except for *Case* 1, no lesions were noted in the *suprarenals*, and in this case there were small punctate hæmorrhages scattered throughout the gland. In turning to the kidney and liver, however, the close similarity of the changes to those seen by Bywaters and Dible (1942), Shaw Dunn, Gillespie, and Niven (1941), and Maegraith and Findlay (1944) was immediately noted.

The *liver* showed a central vein necrosis, and in *Cases* 5 and 10 there was increase of reticulin fibres and early fibrosis. The histology of the *kidneys* varied only slightly from one another. In the main, however, the changes were proportional to the length of survival. All cases had widespread necrosis of the tubular epithelium, most marked in the ascending and descending loops of Henle. Loss of nuclear staining and crenation of the tubular epithelium lying free from the basement membrane were frequently seen without definite cast formation. In later cases epithelial ' plication ', concentrated in the collecting tubules, was also observed. In some, degenerate epithelium was seen lying in the lumen, whilst regeneration of the basement membrane was seen surrounding it.

Alteration in the glomeruli was minimal. Many appeared bloodless and a thin layer of eosinophilic material was arranged peripherally around Bowman's capsule. In the later cases this was not so noticeable. The first convoluted tubules showed only mild degenerative changes. There was a loss of nuclear staining and pyknosis of the muscle. Lying in the lumen of the tubule a ' catarrh ' composed of albuminous material was seen. In the second convoluted tubules changes were more advanced, epithelial degeneration and cast formation being obvious.

The presence of casts was most striking in the ascending and descending limbs of Henle. The numbers varied from case to case and from area to area. The interesting feature was the obvious change in their composition (*Fig.* 401). In those cases dying early they appeared to consist largely of hyaline material; in the later cases pigment casts were seen more frequently. In some, pigment was seen surrounding some central focus, e.g., a fragment of degenerate epithelium or sulphanilamide crystals (*see Fig.* 402). When stained by Masson or Mallory the casts appeared to consist of protein, but the stain

FIG. 401.—Section of the kidney showing degeneration of the epithelium in the ascending and descending limbs of Henle as the most marked feature of this syndrome. The wide variety of cast formation is also noticeable. H. & E. (× 90.)

was taken up irregularly and varied from blue to red. In places granular material was also seen, staining pink with Mallory. At the same time, stripping up of the degenerative epithelium is seen (' plication '—Bywaters and Dible, 1942), whilst the remaining

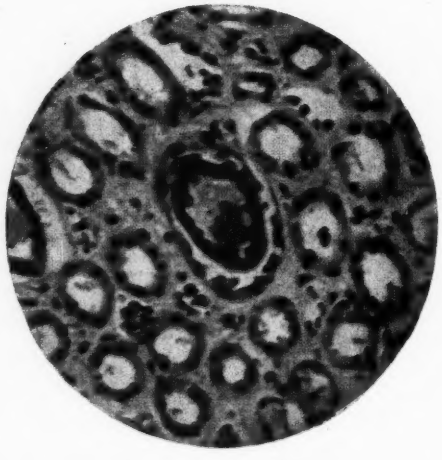


FIG. 402.—The formation of the cast frequently occurs around sulphanilamide crystals. There is also degeneration of the surrounding epithelium. H. & E. (× 185.)

casts were infiltrated with polymorphonuclear leucocytes and plasma cells. This was most marked in the collecting tubules.

All cases showed tubulovenous ruptures (Shaw Dunn, Gillespie, and Niven, 1941; Bywaters and Dible, 1942; Maegraith and Findlay, 1944). In some cases these were obvious, but in other cases were difficult to find. Serial section in areas where interstitial blood was seen led to their discovery in all cases (*Fig.* 403). Tubular ruptures occurred

most frequently in the boundary zone, and, although usually in venous spaces, it also occurred in the interstitial tissues. In some veins, particularly in the later cases, organizing thrombi were also observed (*Fig.* 404).

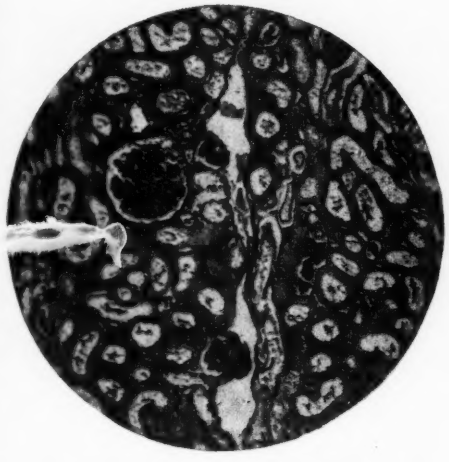


FIG. 403.—The absence of blood from the glomerulus and the 'catarrh' lying within the lumen of the first convoluted tubules is a feature of the syndrome. In addition, two tubules about to rupture into a vein are also seen. The lower has an ante-mortem thrombus adhering to its projecting surface. Mallory. (× 52.)

Serial section of these veins show that they do not necessarily occur as a result of tubulovenous ruptures.

In the interstitial tissue a patchy œdema was frequently observed in early cases. Later cases

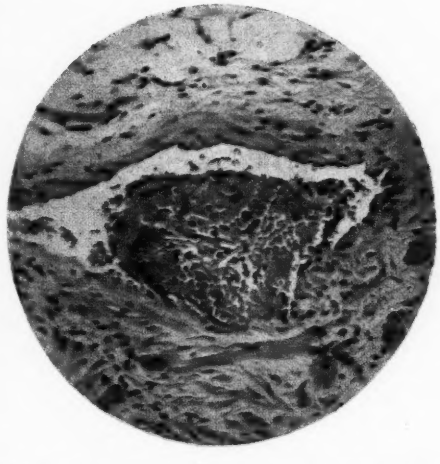


FIG. 404.—An organizing thrombus is seen lying within and attached to the walls of a vein. These are not necessarily associated with tubulovenous ruptures. H. & E. (× 90.)

showed localized groups of histiocytes, and lymphocytes, with early fibrous tissue formation. These were observed more commonly in the boundary zone, and are probably the result of tubulovenous ruptures.

DISCUSSION

Some difficulty was experienced in assessing the part played by the disturbance of the electrolyte balance in the production of traumatic uræmia. Acidosis has long been recognized as accompanying severe clostridial infection (Almroth Wright, 1917), but as the result of the experiences of the Staff of R.A.F. Hospital, Wroughton, it was soon clear that hypochloræmia and acidosis were often present in cases not complicated by gas gangrene. These findings are supported by Cooke et al. (1945). In these patients the response to treatment was gratifying, and, provided saline was given by mouth, in the majority of severely wounded men no other treatment was necessary. It was also noticed that in both these types of cases, in spite of the severity of their biochemical upset, kidney function was not grossly impaired. In support of this, the following case is quoted.

Sgt. P. developed fulminating gas myositis, with acidosis and hypochloræmia. He was aged 23, and was admitted eighteen hours after wounding, under the care of Sq./Ldr. Corson. His injuries consisted of gunshot-wound compound fracture of right femur. A local débridement had been carried out soon after wounding, and he had been evacuated in a Tobruk plaster.

On admission he was extremely restless and complained of pain in his leg. During the night his condition became very much worse and it was clear that he had developed fulminating gas myositis. Emergency amputation was carried out above the fracture site, at the same time transfusion with 3 pints of blood was given, with 120,000 units of polyvalent anti-gas-gangrene serum intravenously. This was followed by 500 c.c. of $\frac{1}{6}$-mol sodium lactate and 1000 c.c. of isotonic saline. A full course of penicillin, 15,000 units three-hourly, was also started. After this he rapidly improved, and was evacuated to an E.M.S. hospital on the sixteenth day.

The biochemical changes in the above case are illustrated in *Fig.* 405, and besides stressing the gross disturbance of electrolyte balance and absence of kidney failure, bring out several other points: (1) There is an immediate alteration in the CO_2-combining power by the intravenous use of 500 c.c. of -mol. sodium lactate solution, but the response to sodium chloride is slow; (2) The body conserves its stock of sodium chloride after depletion, for even after the blood-chloride has returned to the normal there is no secretion of chloride in the urine for three to four days.

It is obvious that adequate biochemical facilities are necessary before treatment of these cases can be undertaken, so that the CO_2-combining power can be observed during treatment. The fact that an acid urine may be secreted in the presence of alkalosis has been noted again and again. This is in accordance with the findings of a large number of workers, and was a feature in the salt-deficiency experiments of McCance and Widdowson (1936). Observations on the urinary *p*H alone may lead to over-alkalinization, and this is not without its dangers. Many writers (Cooke, 1932; McCance and Widdowson, 1937; Oakley, 1935; Cope, 1936; Nicol, 1940; Maegraith and Havard, 1944; and the Army Malarial Research Unit, 1945,) have laid stress on this point. Again, Wakeman et al. (1932) and Maegraith and Findlay (1944) have shown that uræmia in blackwater fever may occur equally in acidosis or in alkalosis (and the kidney lesion in blackwater fever is essentially similar to the kidney lesions in traumatic uræmia). Bywaters (1945) suggested that the

production of an alkaline urine is an essential part of the treatment of cases of traumatic anuria following crush. In this series of cases it was found difficult or impractable to produce an alkaline urine, and it is submitted that this method of treatment is impossible to achieve. Indeed, in view of the ill effects of over-alkalinization, it is probably dangerous. The general survey of the present series and the above cases in the literature would seem to indicate that it is more important to correct the salt deficiency than the alkali reserve. But since a low alkali reserve and low blood-chlorides have been found in other wounded men, it is felt that the electrolyte balance does not, in fact, play a major part in the production of traumatic uræmia.

Turning to the discussion of other possible aetiological factors in production of this type of uræmia, it is well to decide which of the lesions observed in the kidney can be considered as truly characteristic. First, it would appear that the presence of pigment casts in the tubules is not an essential feature. Two additional cases may be referred to in evidence of this. In the first case, epithelial degeneration in the descending and ascending limbs of the loops of Henle and tubulovenous ruptures were present without any casts being found, and the patient died with a raised blood-urea.

Case 13.—A corporal, aged 23, was wounded on Sept. 23, 1944, in Northern Europe. He received a compound fracture of tibia and fibula, with a penetrating wound of his right loin. On the following day an exploratory laparotomy was performed, but, although a large retroperitoneal hæmatoma was found, no perforation of the gut was seen. Twelve days after his injury the wound broke down, discharging duodenal fluid. He was evacuated by air, and, on arrival 20 days after wounding, the wound was still discharging. His blood-urea was 91 mg. per cent and serum-chlorides 303 mg. per cent. During the next ten days three attempts to close the duodenal fistula failed, and, in spite of intravenous and intrasternal infusion of casein hydrolysate, glucose, and saline, he developed uræmia and died, 38 days after initial injury. His blood-urea rose to 290 mg. per cent the day before death.

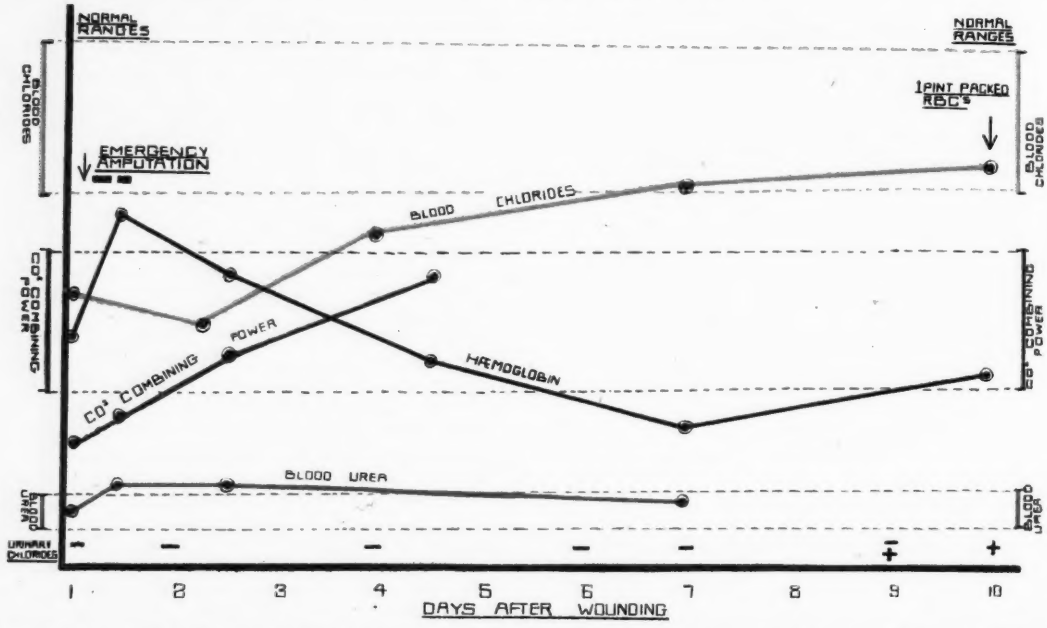


FIG. 405.—Shows the biochemical changes in a seriously wounded man. Note the initial serious reduction in serum CO_2-combining power and in blood-chlorides. Chlorides were not found in the urine until the blood-chlorides had reached normal for three days. This case showed slight elevation of the blood-urea.

At post-mortem examination a large abscess was walled off in the duodenal area. This extended backwards into the loin. A large fistula was found in the second part of the duodenum posteriorly about ¾ in. by 1½ in. in diameter, through which duodenal fluid escaped. The liver showed an early fatty change. The kidneys were enlarged and the cut surface presented the picture of 'traumatic uræmia'. However, on section, although epithelial degeneration of loops of Henle were noticeable, cast formation was conspicuous by its absence (*Fig.* 406). A few tubulovascular ruptures were also found.

FIG. 406.—Marked degeneration of the epithelium of the limbs of Henle are seen without cast formation in a case dying of duodenal fistula, with severe urea retention. H. & E. (× 52.)

This should be compared with that from another case of traumatic uræmia (*Case* 9, *Fig.* 401).

The second case died nine days after a head injury, and the blood-urea was only 56 mg. per cent twenty-four hours previous to death. There were abundant casts in the tubules, but no demonstrable changes in the tubular epithelium.

Sgt. R., was involved in a jeep accident, receiving a fracture of his femur and base of the skull. He was given a blood transfusion and his femur placed in a Tobruk plaster. Forty-eight hours later he was evacuated by air to Wroughton. On arrival, he was semi-conscious. Hæmoglobin was 10·5 g., hæmatocrit 31, alkali reserve 39 vol. per cent. Urine showed no fat. He was treated by oral saline drip, but his condition deteriorated and he died eight days after injury, his blood-urea being 56 mg. per cent one day before death.

Post-mortem examination showed a fracture of the skull extending from the left parietal bone through the petrous bone and across the body of the sphenoid, extending forward through the wing of the sphenoid on the right side. Considerable œdema and laceration of the brain was noticeable. No evidence of fat embolism was seen. No other abnormality of the other organs was seen except that the lungs were extremely œdematous. On section, the kidneys did not show noticeable epithelial tubular changes, although large numbers of casts were seen (*Fig.* 407).

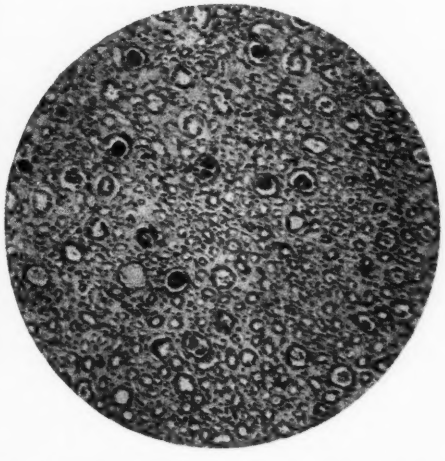


FIG. 407.—Cast formation in the collecting tubules and limbs of Henle of the kidney are seen in a case dying from head injury, and without severe urea retention. H. & E. (× 52.)

Cumings (1942) has reported similar findings.

Taking these two cases with the previously described cases of traumatic uræmia, it can be stated that pigment casts may be present without evidence of renal failure and that all cases with degeneration of the loops of Henle show evidence of renal failure. It would appear that the tubular change is the essential and characteristic lesion in these cases dying of uræmia and that the presence of pigment casts is incidental. This is in accordance with the opinion expressed by De Navasquez (1940), Bywaters and Dible (1942), Eggleton et al. (1943), and Maegraith and Findlay (1944), who did not accept the original theory put forward by Barratt and Yorks (1909), Baker and Dodds (1925), and Morrison (1941) that renal failure was caused by a blockage of the tubules by epithelial debris and casts.

The possible causes of this tubular degeneration must now be discussed. Many workers have suggested that some toxic substance acts upon the renal tubular epithelium (Minami, 1923; Bywaters and Beale, 1941; Bywaters and Dible, 1942; and Eggleton, 1944). It has been suggested that this nephrotoxic substance is derived from damaged muscle, but in this series now reported many of the cases had little muscle damage, and during the same period many wounded men with extensive muscle necrosis were examined and showed little, if any, disturbance of renal function. Experimental workers have been unable to reproduce the syndrome by the crushing of limbs (Bywaters and Popyak, 1942; Duncan and Blalock, 1942; and Eggleton et el., 1943), nor is it produced by the introduction of pigment into the circulation, unless the animal is in a state of acidosis (Bywaters and Stead, 1944; Bing, 1944). Foy et al. (1943), in criticizing previous workers in this field, suggest that dehydration and the substance used to produce acidosis have an equally important role in the production of renal failure.

The views of Tombs (1942) and Maegraith, Havard, and Parsons (1945) have recently attracted attention. These authors have suggested that there are many examples of this syndrome in the literature, and that a diminution in blood-flow through the kidney is the possible factor in its production. They believe that the renal anoxia produced in this way, should it persist, would cause damage in the areas last supplied by blood, i.e., the loops of Henle, the area in which the maximal damage is found. The work of Cournard et al. (1943) and Lauson et al. (1944) has brought conclusive proof that vaso-constriction of renal arteries occurs in shock. When shock is produced in animals by bleeding, uræmia may develop (Meyler, 1935; Corcoran and Page, 1943; Phillips et al., 1946). Again, temporary occlusion of the renal vessels with local anoxia produces a picture comparable to traumatic uræmia (Scarff and Keele, 1943; Van Slyke et al., 1944; Badenoch and Darmady, 1947). It must be pointed out that in the present series of cases of traumatic uræmia, factors which might lead to renal vaso-constriction were invariably present (i.e., shock and damage to major vessels and limbs). Barnes and Trueta (1942) have shown that localized trauma of one leg in animals will lead to widespread arterial spasm, which might extend to vessels supplying more vital organs. Trueta (1945) has drawn attention to the fact that in shock over-stimulation of peripheral nerves may cause persistent spasm of renal and other arteries, and this would lead to interference with the blood-supply and result in renal anoxia.

There seemed, therefore, strong evidence that vasospasm was responsible. It followed that should the spasm be relieved sufficiently early the renal circulation might be restored, and the patient recover. For many years it has been realized that renal vasoconstriction can be obtained by stimulation of the splanchnic and peripheral nerves (Bernard, 1859; Bradford, 1889; Homer Smith, 1941), and that division of the nerves prevents this action.

Trueta (1944) suggested that a 'splanchnic block' might act in a similar manner in the human. The value of this manœuvre has been shown in 'reflex anuria' (Rubritius, 1925; Havelick, 1925; Haslinger, 1927), and in incompatible blood transfusion (Hesse and Filatov, 1933; Raymond Peters, 1942). Cubitt (1936) found that a similar effect could be obtained by a high spinal anæsthetic. The effect of splanchnic block was tried in one patient (*Case* 16). This Polish private was wounded on April 25, 1945, receiving a gunshot wound of his left buttock and thigh. On the following day his leg was found to be ischæmic and the femoral artery injured. An immediate amputation was performed through the thigh. His post-operative condition was described as only fair. Five days after wounding he started to hiccup, and on the sixth day he was evacuated by air and admitted to Wroughton. He was now vomiting, and his blood-urea was found to be 500 mg. per cent (*Fig.* 408). His fluid intake was limited, and on the eighth day after wounding his blood-urea had risen to 820 mg. per cent. Splanchnic block was then performed. His blood-pressure fell, and on the ninth day after wounding his blood-urea had fallen to 220 mg. per cent. From this day his condition rapidly improved. He was transferred twenty-seven days after wounding, his blood-urea being 40 mg. per cent on discharge. A further case, *Case* 17, was treated by this method, and, although urinary secretion was re-established, death occurred from cardiac failure. The dramatic improvement seen in *Case* 16 can occur spontaneously (Belsey, 1942); therefore it is unwise, at this stage, to attribute too much to the effects of the splanchnic block. Porritt et al. (1945), in discussing casualties from the B.L.A., have commented unfavourably on this form of treatment for traumatic uræmia. It is pointed out, however, that splanchnic block cannot be successful if irreversible damage has already occurred, and, as Raymond Peters (1942) has said, "It cannot be too urgently stressed that whatever, if any, value splanchnic block may possess, it should only be considered the key for unlocking the door to subsequent judicious treatment."

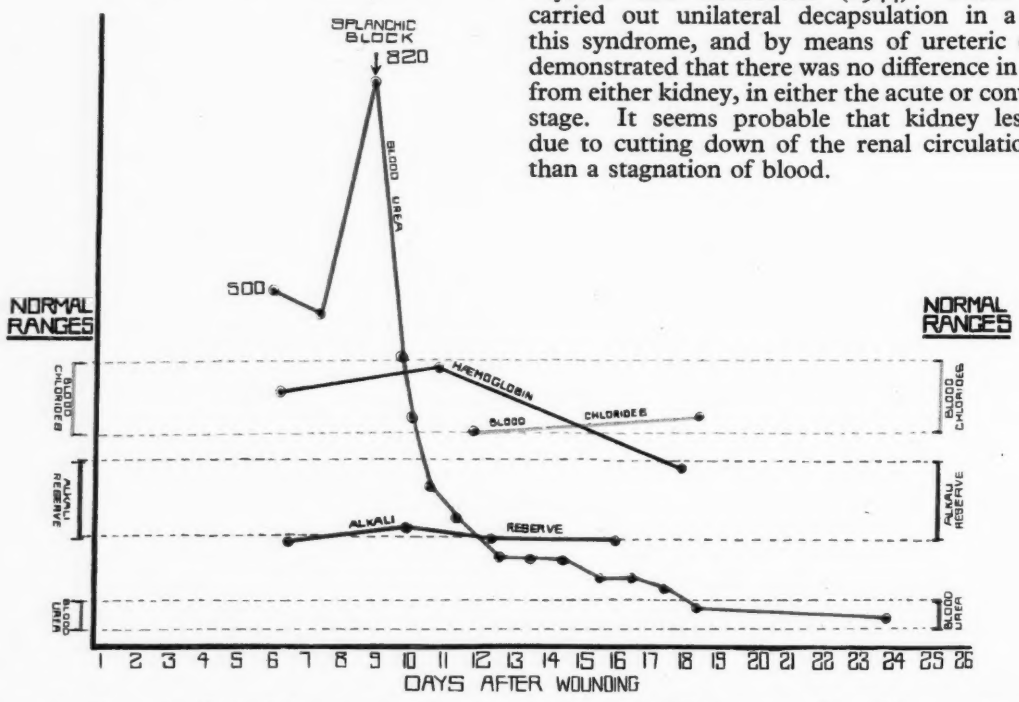

SPLANCHNIC BLOCK
820
500
BLOOD UREA
NORMAL RANGES
NORMAL RANGES
HÆMOGLOBIN
BLOOD CHLORIDES
ALKALI RESERVE
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26
DAYS AFTER WOUNDING

FIG. 408.—Shows the biochemical course of a case of traumatic uræmia with recovery. The striking response to splanchnic block suggests this treatment as a possible new line of therapy.

Earlier in this paper reference has been made to the 'crisis' which is a feature of those cases with recovery. The theory of vasospasm and its sudden relief can satisfactorily explain their rapid improvement, and is seen to best advantage in *Case* 16. It would also account for the fact that kidney function tests performed in the convalescent stage show no residual damage.

Peters (1945) has recently suggested that intra-renal pressure is responsible for the degeneration of the tubules. He suggests that it occurs as the result of stagnation of blood, and the condition can be relieved by decapsulation of the kidneys. This seems to be a heroic measure to undertake in a patient desperately ill, particularly in view of the work of Styrons and Leadbetter (1944). These workers carried out unilateral decapsulation in a case of this syndrome, and by means of ureteric catheters demonstrated that there was no difference in function from either kidney, in either the acute or convalescent stage. It seems probable that kidney lesions are due to cutting down of the renal circulation rather than a stagnation of blood.

In conclusion, there are many conditions which are followed by a similar syndrome, often terminating in fatal uræmia (Bywaters and Dible, 1942; Foy et al., 1943; Maegraith, Havard, and Parsons, 1945). This syndrome is essentially the same as that now described as occurring in battle casualties, and the same renal lesion, degenerate changes in the loops of Henle, is present in all. A satisfactory explanation of the cause has not yet been given. The most acceptable theory seems to be that of renal anoxia, which may well be brought about by vasospasm. This in its turn may be produced by various conditions such as shock, sensitizing antigens,

dehydration, and hypochloræmia. It is suggested that vasospasm is the immediate cause of the renal failure and tubular damage, and further experimental work is essential to test this hypothesis.

SUMMARY

1. A syndrome of uræmia following the hazards of war based on 17 cases is described with special reference to biochemical and histological data.

2. The aetiological factors and the similarity to other conditions have led to the conclusion that the ultimate cause of death is renal anoxia. The mechanism of the production of renal anoxia by alteration of the blood-supply to the kidney by vasospasm is debated.

3. The dangers of over-alkalinization, and possible help through splanchnic block, are brought forward.

My thanks are due to Air Marshal Sir Harold Whittingham, K.C.B., K.B.E., Director-General Medical Services, for his continual interest, and for permission to publish this paper.

Also to Group Captains J. Russell and G. A. M. Knight, Officers Commanding this Royal Air Force Hospital.

I would also record my indebtedness to Professor E. J. King, of the Post-graduate School, for carrying out certain of the biochemical investigations on behalf of the Medical Research Council, and to Mr. Hennell, of the Metal Box Company, for the coloured microphotographs.

It is with the greatest pleasure that I record my grateful thanks to my medical colleagues, sisters, nursing orderlies, and laboratory staff, for without their whole-hearted co-operation the collection of this data could not have been possible.

I would also like to express my thanks to L.A.C. W. Donnelly and Corporal Hagger for drawing the charts, and for tabulating the many case records of this and allied conditions.

BIBLIOGRAPHY

ARMY MALARIAL RESEARCH UNIT (1945), *Lancet*, **2**, 701.
BADENOCH, A. W., and DARMADY, E. M. (1947), *J. Path. Bact.* [*To be published.*]
BAKER, S. L., and DODDS, E. C. (1925), *Brit. J. exp. Path.*, **6**, 247.
BARNES, J. M., and TRUETA, J. (1942), *Brit. J. Surg.*, **117**, 74.
BARRATT, J. O. W., and YORKE, W. (1909), *Ann. trop. Med.*, **3**, 1.
BELSEY, R. H. R. (1942), *Proc. R. Soc. Med.*, **35**, 328.
BERNARD, CLAUDE (1859), *Leçons sur les Proprietes physiologiques et les Alterations pathologiques des Liquides de l'Organisme*, **2**, 162. Paris.
BING, R. J. (1944), *Bull. Johns Hopk. Hosp.*, **74**, 161.
BRADFORD, J. R. (1889), *J. Physiol.*, **10**, 358.
BYWATERS, E. G. L. (1945), *Brit. med. Bull.*, **3**, 694, 107.
— — and BEALL, D., (1941), *Brit. med. J.*, **1**, 427.
— — and DIBLE, J. H. (1942), *J. Path. Bact.*, **54**, 3.
— — and POPYAK, G. (1942), *Surg. Gynec. Obstet.*, **75**, 622.
— — and STEAD, J. K. (1944), *Quart. J. exp. Physiol.*, **33**, 53.
COOKE, A. M. (1932), *Quart. J. Med.*, **25**, 527.
COOKE, W. T., FRASER, A. C., PEENEY, A. L. P., GOVAN, A. D. T., BARLING, S. G., GARFIELD THOMAS, LEATHER, J. B., ELKES, J. J., and MASON, R. P. SCOTT. (1945), *Lancet*, **1**, 487.
COPE, C. L. (1936), *Brit. med. J.*, **2**, 914.
COCORAN, A. C., and PAGE, I. H. (1943), *J. exp. Med.*, **78**, 205.
COURNARD, A., RILEY, R. L., BRADLEY, S. E., BREED, E. S., NOBLE, R. T., LAUSON, H. D., GREGERSON, M. I., and RICHARDS, D. W. (1943), *Surgery*, **13**, 964.
CUBITT, A. W. (1936), *Brit. J. Surg.*, **24**, 215.
CUMINGS, J. N. (1942), *J. Path. Bact.*, **54**, 532.
— — (1942), *J. Neurol. Psychiat.*, **5**, 40.
DARMADY, E. M., SIDDONS, A. H. M., CORSON, T. C., LANGTON, C. D., VITEK, Z., BADENOCH, A. W., and SCOTT, J. C. (1944), *Lancet*, **2**, 809.
— — (1946), "The Traumatic Uræmia Syndrome", M.D. Thesis, Cambridge University.
DE NAVASQUEZ, S. (1940), *J. Path. Bact.*, **51**, 413.
DUNCAN, G. W., and BLALOCK, A. (1942), *Ann. Surg.*, **115**, 684.
DUNN, J. S., GILLESPIE, M., and NIVEN, J. S. F. (1941), *Lancet*, **2**, 549.
EGGLETON, M. G., RICHARDSON, K. C., SCHILD, H. O., and WINTON, F. R. (1943), *Quart. J. Physiol.*, **32**, 89.
— — (1944), *Lancet*, **2**, 208.
FOY, H., ALTMAN, A., BARNES, H. D., and KONDI, A. (1943), *Ibid.*, **36**, 197.
HASLINGER, K. (1927), *Z. urol. Chir.*, **21**, 174.
HAVLICEK, H. (1925), *Zbl. inn. Med.*, **46**, 465.
HESSE, E., and FILATOV, A. (1933), *Z. ges. exp. Med.* **86**, 211.
LAUSON, H. D., BRADLEY, S. E., COURNARD, A., and ANDREWS, V. V. (1944), *J. Clin. Invest.*, **23**, 381.
MAEGRAITH, B. G., and FINDLAY, G. M. (1944), *Lancet*, **2**, 403.
— — and HAVARD, R. E. (1944), *Ibid.*, **2**, 338.
— — — — and PARSONS, D. S. (1945), *Ibid.*, **2**, 293.
MCCANCE, R. A. (1936), *Lancet*, **1**, 643.
— — and WIDDOWSON, E. M. (1936), *Proc. roy. Soc.*, s. B., **120**, 228.
— — — — (1937), *Lancet*, **2**, 247.
MEYLER, L. (1935), *Acta med. scand.*, **87**, 313.
MINAMI, S. (1923), *Virchows Arch.*, **245**, 247.
MORISON, J. E. (1941), *J. Path. Bact.*, **53**, 403.
NICOL, B. M. (1940), *Quart. J. Med.*, **9**, 91.
OAKELEY, W. (1935), *Lancet*, **2**, 187.
PETERS, H. RAYMOND (1942), *Ann. int. Med.*, **16**, 547.
PETERS, J. H. (1945), *Ibid.*, **23**, 222.
PHILLIPS, R. A., DOLE, V. P., HAMILTON, P. B., EMERSON, K., ARCHIBALD, R. M., and VAN SLYKE, D. D. (1946), *Amer. J. Physiol.*, **145**, 314.
PORRITT, A. E., DEBENHAM, R. K., and ROSS, C. C. (1945), *Brit. med. J.*, **2**, 377.
RUBRITIUS, H. (1925), *Wien. klin. Wschr.*, **38**, 749.
SCARFF, R. W., and KEELE, C. A. (1943), *Brit. J. exp. Path.*, **24**, 147.
SMITH, H. (1941), Harvey Lectures, 35.
STYRON, C., and LEADBETTER, W. (1944), *New Engl. med. J.*, **230**, 721.
TOMBS, J. W. (1942), *J. trop. Med. (Hyg.)*, **45**, 125.
TRUETA, J. (1944), personal communication.
— — (1945), *Lancet*, **2**, 415.
VAN SLYKE, D. D., PHILLIPS, R. A., HAMILTON, P. B., ARCHIBALD, R. M., DOLE, V. P., and EMERSON, K. (1944), *Trans. Ass. Amer. Phys.*, **58**, 119.
WAKEMAN, A. M., MORRELL, C. A., EISENMAN, A. J., SPRINT, D. L., and PETERS, J. P. (1932), *Amer. J. trop. Med.*, **12**, 407.
WRIGHT, A. E. (1917), *Lancet*, **1**, 1.

OSTEITIS PUBIS AFTER SUPRAPUBIC OPERATIONS ON THE BLADDER
WITH A REPORT OF TEN CASES

By E. KIRZ
NORTH MIDDLESEX COUNTY HOSPITAL

In the second quarter of the twentieth century endoscopic operations have more and more supplanted the suprapubic approach to the bladder. There will, however, always remain many indications for the latter method and possible complications cannot be disregarded.

Osteitis pubis is mentioned in very few of the published papers dealing with complications of suprapubic operations on the bladder. The standard text-books on urology and general surgery have practically no reference to this condition. Two exceptions are Hugh Cabot's *Modern Urology* (1936) and Lichtenberg's *Handbuch der Urologie* (1926). H. Cabot gives a very good account, while in Lichtenberg's work, F. Voelcker and H. Boeminghaus devote one short sentence to "osteomyelitis after suprapubic vesical operations".

The complication was originally described by Dr. Edwin Beer (1926) and for many years was thought to occur only after incision into the bladder. In 1933, however, Barnes reported a case after a perineal prostatectomy. Wilensky (1938) called attention to 2 cases in women: the first followed symphysiotomy during a difficult labour, and in the other a vaginal discharge which appeared after "normal" delivery was traced to a vaginal sinus leading to the pubic bone. Kleinberg (1942) reported a third case in a woman, but admits "it is difficult to judge how the pubes became infected . . . it was certainly not by direct extension of an adjacent infectious process". Strictly speaking, therefore, this last case does not fit into this group of traumatic osteitis. However, he also states that "instances of typical pubic osteitis have followed a hernia operation which was infected, a prostatic abscess, and an occupational trauma to the lower abdomen".

Having recently recognized a case of osteitis pubis, I have inquired into the subsequent history of 300 cases upon which suprapubic operations on the bladder had been performed during the past three years. At the time of writing 103 patients were no longer alive and 23 were not traceable. Among the remaining 174 patients 6 cases of osteitis were verified, and, in addition to these, 3 others have been observed during the last few months. Only those cases were taken into account in which pre-operative X-ray films were still available for comparison with those taken two or three years after operation. In this series the incidence of osteitis pubis was 3 per cent, and it is clear that this serious and as yet obscure complication is by no means a rare one, and it is likely that minor degrees of the trouble are frequently overlooked.

CASE REPORTS

Owing to the limitations of space only the positive findings are given.

Case 1.*—W. W., aged 62. Harris prostatectomy for a simple adenoma on Oct. 30, 1943, and discharge home on Nov. 23.

He was seen again on Dec. 22. In the meantime micturition had been free, but there was slight terminal dysuria. Since about the tenth day after discharge from hospital he had had pain in the area of the symphysis pubis, along the inner aspect of both thighs, and over both tubera ischii, preventing him from properly sitting down. On examination he was extremely tender over these areas. The adjoining muscles were all spastic.

During the following months the above symptoms got so bad that he could only with great effort sit down, get up, or move about in bed. At times he was entirely incapacitated and found relief only by complete rest in bed. In February, 1944, there was great wasting both of the glutei and of the adductors. Very slowly his symptoms subsided, but were not gone until November, 1944. An X-ray film taken in September, 1944, showed new-bone formation in the inferior rami and irregular outlines of the medial borders of the pubic bone, with a considerable gap at the site of the symphysis. (*Fig.* 409.)

Case 2.—A. J. Th., aged 58. On March 31, 1945, prostatectomy for a clinically benign adenoma. Convalescence delayed by right upper-lobe pneumonia. Microscopy of the prostate showed early malignant changes, but there were no further complications and patient was discharged home on April 28.

He was readmitted on May 10 because he had symptoms and signs of osteitis pubis. Digital examination also revealed a small symptomless carcinoma of the rectum which was fixed to the lower sacrum. The temperature was 100·2° F. There was occasional discharge of pus from the lower end of the suprapubic wound, but bare bone could never be felt with a probe. The symptoms gradually subsided and he was up and about again in October, 1945. Earlier in June left inguinal colostomy had been performed.

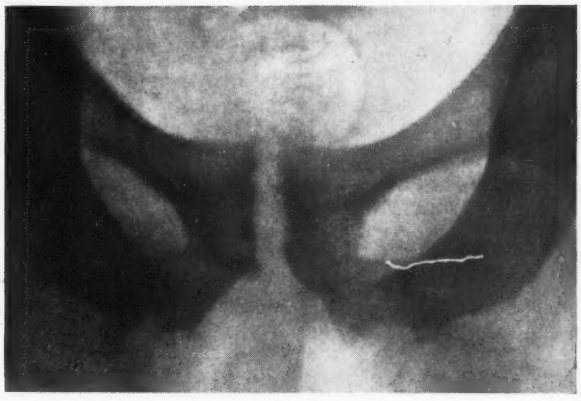
On Nov 10, a recently formed vesical calculus had to be removed, but before proceeding with this an incision was made from the inferior pubic angle along the right ramus to the right ischial tuberosity. On cutting down to bone across parts of the adductor magnus origin about 10 c.c. of pus welled up. Specimens were taken of thickened connective tissue and rough bone and sent with the pus for pathological examination. The following cystolithotomy showed the neck of the bladder to be soundly healed and there was no evidence of any recurrent malignant changes.

The pathologist (Mr. T. H. C. Benians) reported as follows:—

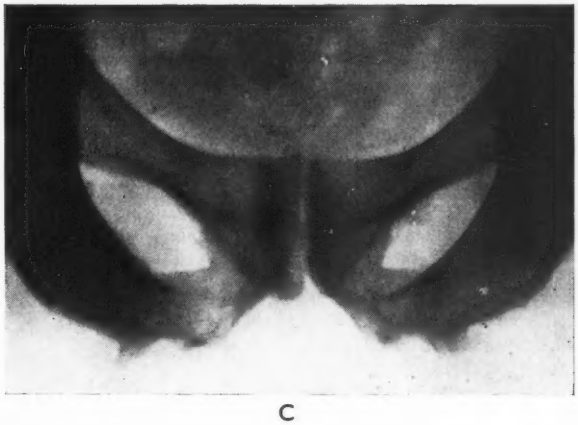
"1. Tissue taken from the wall of the abscess cavity lying over the inner eroded surface of the inferior pubic ramus consists of pyogenic granulation tissue and fatty tissue showing chronic inflammation and fibrosis. The granulation tissue shows some rather unusual features in the form of nodules and sheets of epithelioid cells and giant cells somewhat suggestive of a tuberculous process, although nests of polymorphs constituting small abscesses occur. It is undoubtedly a picture of a chronic pyogenic infection. A few small spicules of bone disintegrated by the microtome are present.

* This case was shown at the Meeting of the Middlesex County Medical Society on June 9, 1945.

A



B



C

FIG. 409.—A, Before operation. Normal pelvic bones. B, 11 months after operation. Still showing diffuse absorption, fraying at the symphysis, but also new-bone formation at both inferior rami. C, 26 months after operation. Advanced signs of healing (clinically symptomless).

"2. A fragment of spongy bone of which the periosteal surface does not appear in the sections. It is composed of trabeculæ of original lamellar bone and the fatty marrow shows a mild degree of chronic inflammation.

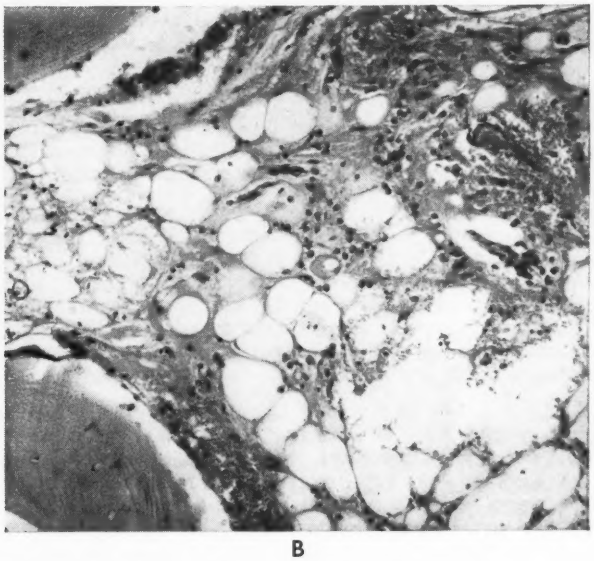
"3. 5 c.c. of pus. Films show degenerate pus, but no organisms were seen. Cultures produced a scanty growth of *Ps. pyocyanea.*

"It is a fair assumption that the inflammation of the pubic bone and the suppuration of the adjacent soft tissues are associated processes. Both are of a mild or subacute type, the organism isolated, i.e., *Ps. pyocyanea,* is not uncommonly found in old-standing infections of the bladder, although usually accompanied by staphylococci, and the infection is probably derived from this source. Perhaps a staphylococcus has taken part in the inflammatory process and has since died out.

"Such low-grade infecting organisms as these might well be associated with inflammations of the chronic type described here." (*Figs.* 410, 411.)

Case 3.—J. H. W., aged 45. Partial cystectomy for carcinoma of the bladder on June 20, 1945. Three weeks later patient was passing most of his water by the urethra. From the fourteenth post-operative day, however, the temperature was slightly raised, and from the twenty-first day he complained that movement of his pelvis caused pain over the symphysis, radiating down the inner aspect of the right thigh. By August the full picture had developed, including terminal dysuria, pain on defæcation, and wasting of the adductor muscles.

A



B

FIG. 410.—Fatty marrow with chronic inflammatory infiltration, mainly plasma cells. (A, × 28; B, × 117.)

Later radiographs clearly showed periosteal new-bone formation, with eventual reappearance of normal bone. He is back at his old job since January, 1946.

Case 4.—J. F. E., aged 71. March 13, 1945: One-stage prostatectomy for a benign adenoma. Discharged on March 28. He had to be readmitted for bilateral epididymitis three days later.

Slight fever and symptoms of osteitis appeared first on June 5. Radiographs showed small areas of absorption in the lower third of the pubic body and in the inferior rami. New-bone formation in both inferior rami was seen in December, 1945.

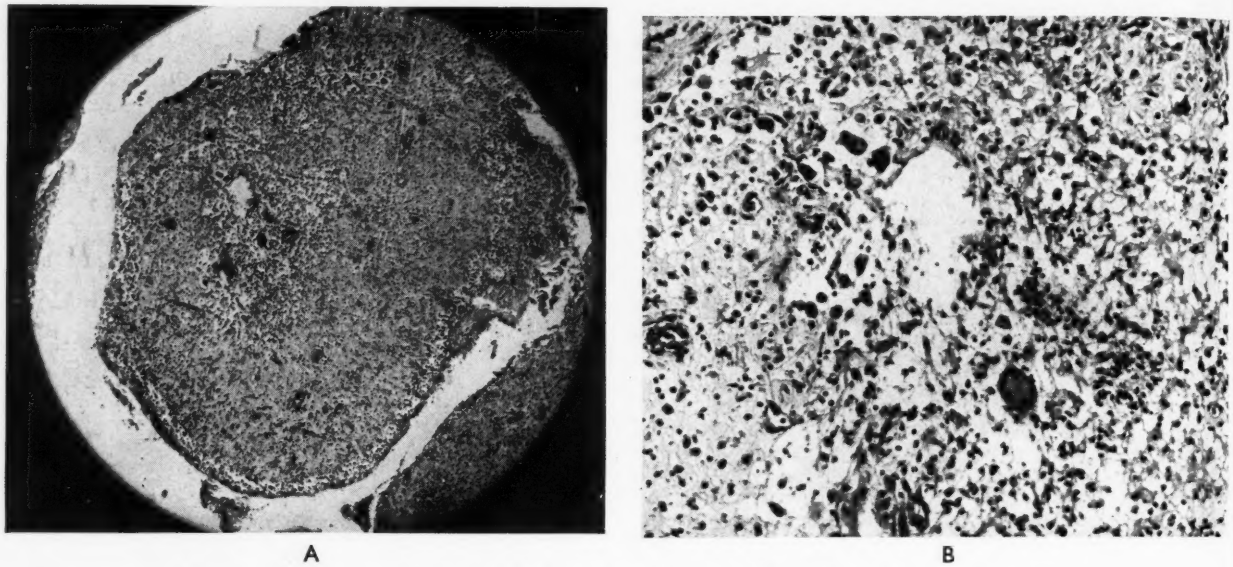
Case 5.—E. A. Th., aged 65. Feb. 28, 1943: Suprapubic cystostomy for acute retention due to fibrosis of the bladder neck. He attended the out-patient department at intervals to have his de Pezzer catheter changed. During this time he had persistent symptoms of osteitis pubis, which were, however, not recognized then, and had again subsided by July, 1945. X-ray films taken now showed unmistakable new-bone formation over the inferior rami and lower borders of pubic body.

Case 6.—D. P., aged 75. May 22, 1945: Suprapubic cystostomy for acute retention due to a clinically benign enlargement of the prostate. Symptoms and sings of osteitis pubis appeared first towards the end of June, 1943, but were not appreciated until radiographs taken in November, 1945, proved the case.

Case 7.—W. S. J., aged 79. Aug. 14, 1943: Harris prostatectomy for a simple adenoma. Pain over the symphysis, adductor origins, and over the ischial tuberosities appeared first in mid-September, 1943. Two years later, although symptomless, patient was still tender over the above-mentioned areas and X-ray films showed changes due to osteitis, particularly of the left pubis and inferior ramus.

Case 8.—A. R. B., aged 68. Sept. 8, 1943: Harris prostatectomy for a cystic adenoma. Apart from left epididymitis, recovery was uneventful and he was discharged home on Sept. 24, 1943. A few weeks later his symptoms were thought to be due to an inflammation of the vas. In September, 1945, he was still tender over the symphysis and left superior ramus and X-ray films showed three areas of residual opacities in the left corpus pubis, with streaks of calcification across the symphysis.

Case 9.—A. Th., aged 75. June 21, 1944: Harris prostatectomy for a simple adenoma. Discharged on July 4, 1944. First symptoms four weeks later. Mid-September, 1945, he was still found to be tender over the sites in question and X-ray films showed diffuse areas of absorption at the medial portions of the pubic bodies, more obvious on the right.



A B

FIG. 411.—Pyogenic membrane showing giant cells and nests of polymorphs. (A, × 34; B, × 117.)

Case 10.—A. G., aged 79. Nov. 25, 1942: Freyer's prostatectomy for fibromuscular hypertrophy. He had been up and about again since the nineteenth post-operative day. From the twenty-third day, however, the temperature was slightly raised and he was complaining of pain over the symphysis and right groin.

Two years later, in the course of this follow-up, osteitis pubis was recognized and confirmed by X rays.

CLINICAL FEATURES

The symptoms, the physical signs, and the X-ray appearances of this condition are to be found in the literature, but as they seem to have largely passed unnoticed, it may not be superfluous to repeat them here.

Three to six weeks after operation the patient complains of pain in the region of the symphysis pubis. This pain may extend towards the perineum and radiate down the inner side of one or both thighs. If the man is still in bed the slightest movement of the thighs or attempts to turn over or to sit up will cause pain; if he is up, walking is difficult or impossible. In other cases standing may be easier than sitting, the latter causing unbearable pain over the tubera ischii. Urinary symptoms are rare, but terminal dysuria occurs in cases in which the origin of the perineal muscles is involved; similarly, there may be pain on defæcation. These symptoms may actually make the patient take to his bed again, because only full rest will relieve them. After many months of illness he usually gets well, but a few cases of death from severe sepsis have been reported.

On examination, the areas mentioned are extremely tender to touch. All active and passive movements of muscles originating from that part of the pelvis involved by the osteitis are limited and very painful. These muscles are at first spastic; later, in severe cases, they may show great wasting. At times there may be some discharge of urine or pus from the lower part of the operation scar, but no pathogenic organisms are obtained in culture. This discharge, moreover, does not seem an essential part of the trouble, as it often occurs in cases showing no signs of osteitis pubis. Slight fever at the onset of symptoms is, however, a characteristic feature.

No constant picture can be drawn of the early X-ray changes. Indeed, the radiographs may at first show no change. Later the first signs of rarefaction appear at the site of most trauma. After major operations this area is usually the medial portion of the lower third of one or both corpora pubis. After minor operations, fraying appears first at the upper corners of the pubic bone and in mild cases the condition may be arrested at this stage. Most of the established cases, however, later on show a definite spread involving variable amounts of pubic bone and ischium. Very rarely the condition may extend to the hip-joint, as recently reported by Silver (1941).

The symphysial cartilage may be absorbed, giving the appearance of separation of the symphysis, and finally bony ankylosis may result. Formation of a sequestrum is the exception rather than the rule, but areas of osteoporosis and osteosclerosis are seen later. These areas may be difficult to distinguish from metastatic growth, but reappearance of normal bone, or periosteal new-bone formation resulting in osteophytes and bony spurs, with the disappearance of symptoms, eventually dispel any doubts.

PATHOLOGY

Most writers agreed that the condition was due to infection. Only Wheeler (1941) rejected this and asserted that osteitis pubis is identical with acute bone atrophy (Sudeck) rather than due to infection.

He supported his view with the results of a series of experiments, in which he failed to reproduce periostitis in rabbits after the injection of mixed cultures under the pubic periosteum.* Though it would not be wise to attach too much importance to these animal experiments, it is agreed that in mild cases of osteitis pubis only areas of decalcification can be seen in radiographs. On the other hand, most of the cases show all those bone changes usually associated with infection. Furthermore, the symptoms in typical acute bone atrophy are never relieved by rest, and pyrexia does not occur.

Of the 10 cases reported here, only 1 was explored, partly because of individual circumstances and partly because in the literature mainly non-interference has been advocated.

The findings of pus, granulation tissue, and rough bone in this one case in which the clinical picture and radiography were almost identical with the other 9, however, seemed to prove the case.

PATHOGENESIS

Various explanations have been offered, but two main factors appear to be essential in the production of the infection: (1) Leakage of infected urine into the retropubic space; (2) Trauma both during and after operation.

It is asserted by many urologists that leakage into the retropubic space is, or at least has been, unavoidable. Regarding the second factor, Beer (1928) has suggested that "the infection develops in the periosteum as a result of injury to the periosteum, by traction on the attached rectus muscle". Injury by direct instrumentation, by local anæsthesia, or by pressure of the drainage tube against the underlying tissues, has also been blamed. But it has also been suggested that in other types of cases the infection travels by direct extension to the pubes. Wilensky (1938) says: "the bone is secondarily bared and infected, after the separation from it of the necrotic tissue . . . infection in the osseous tissue is aided by injury to the periosteum".

It is, however, against all surgical conceptions to assume that an infection, particularly a low-grade one that spreads by extension, should do so across fascial planes, instead of along them, unless first of all this path has been laid open.

It is the purpose of this paper to show that trauma inherent in the classical suprapubic approach is the main cause of osteitis pubis, as this approach involves operating as near as possible to the pubic bone.

When dividing the consecutive layers of the abdominal wall, the retromuscular space and the space of Retzius are deliberately opened. (Trauma No. 1.)

In the following steps of the operation, retraction may cause avulsion of fibres of the rectus muscle, or the two layers of the transversalis fascia from the pubic bone. (Trauma No. 2.)

For suprapubic prostatectomy in particular (and similarly in other operations on the bladder) the enucleation of the glandular tissue more often than not involves anything but gentle handling. Though in most cases two fingers in the rectum may sufficiently immobilize the prostate bed, very often, in my opinion, rupture of the puboprostatic ligaments is inevitable. (Trauma No. 3.)

The early symptoms and X-ray findings seem to support the three traumata mentioned.

The factor of infection is obviously essential, but, unless the aforementioned fascial spaces are primarily traumatized and the bone bared from its coverings, it is difficult to see why infection of the pubic bone should occur rather than propagation of the inflammatory process along the fascial planes. Suppuration in these perivesical spaces has been frequently observed after endoscopic operations, but osteitis pubis has never been reported.

The only argument in favour of a process which spreads slowly to the periosteum is the long interval between operation and onset of symptoms. This long interval, however, is only apparent, inasmuch

* Wheeler also mentions a case of Dr. P. Riaboff, which at autopsy showed "no inflammatory processes of the bone or periosteum, but definite atrophy of the cellular elements of the bone". This case was to be reported separately; up to now, however, I have not been able to find a reference to it in the *Quarterly Cumulative Index Medicus*.

as the early slight symptoms are naturally put down to unavoidable post-operative pain. Only later, after fully established involvement of the adjacent tissues (origin of muscles), will the symptoms become manifest.

Severe sepsis, toxic nephritis, and anuria during the immediate post-operative period are admittedly rare complications. It would, however, be interesting to know how many of these unfortunate complications could have been avoided if the prostatic plexus had not been injured and infected at operation, and also how many cases would have developed osteitis pubis had they not succumbed to the more severe infection.

It would seem that the opening of the retromuscular space and of the space of Retzius can be eliminated if a technique slightly modified from that recently described by Dodson (1944) were adopted. According to this, the separation of the recti and pyramidalis muscles is carried out in the usual manner. Then, about 3 cm. above the level of the symphysis a 2-in. long transverse incision is made through the anterior and posterior layer of the (continuation of the) transversalis fascia, prevesical fat, and vesical layer of the pelvic fascia. The lower flap is then dissected downwards off the anterior bladder wall for about 4 cm. and approximated with two or three interrupted sutures to muscle at the lower angle of the wound. The two spaces are thereby securely shut off.

In a certain number of cases, as already admitted, it may be impossible not to injure the puboprostatic ligaments. In most cases, however, it should be avoidable, particularly if this complication is kept in mind.

SUMMARY

Osteitis pubis, after suprapubic operations on the bladder, appears to be a clinical entity. After 300 such operations 10 cases have been discovered and are reported here. Symptoms, physical signs, and X-ray findings are described and the aetiology discussed, emphasizing the factor of operative trauma in the operation as currently practised. A method of operation to minimize this factor is suggested.

I am indebted to Mr. Ivor Lewis for permission to publish this paper, and I wish to express my gratitude to him and Mr. F. J. F. Barrington, who read the manuscript, and Mr. T. H. C. Benians for their invaluable suggestions.

My thanks are also due to Mr. E. V. Willmott, A.R.P.S., for preparing the radiographs and microphotographs.

REFERENCES

ASCHER, P. W. (1924), *J. Urol.*, **12**, 251.
BARNES, F. (1933), *Texas St. J. Med.*, **28**, 601.
BEER, E. (1928), *J. Urol.*, **20**, 233.
— — (1924), *Int. J. Med. Surg.*, May.
CABOT, H. (1936), *Modern Urology*, **11**, 164.
DODSON, A. I. (1944), *Urological Surgery*.
GOLDSTEIN, A. E., and ABESHOUSE, B. S. (1929), *Surg. Gynec. Obstet.*, **49**, 477.
— — and FERBER, W. L. F. (1938), *J. Mt Sinai Hosp.*, **4**, 1052.
KINLOCK, R. E., and DRECHSLER, L. S. (1930), *Long Is. med. J.*, **24**, 335.
KLEINBERG, S. (1942), *J. Urol.*, **48**, 635.
KRETSCHMER, H. L. (1930), *Ibid.*, **23**, 573.
LICHTENBERG, A. (1926), *Handbuch der Urologie*, **1**, 109.
LAZARUS, J. A. (1936), *Ann. Surg.*, **103**, 310.
MATHÉ, C. P. (1940), *J. Urol.*, **43**, 543.
SILVER, C. (1941), *Bull. Hosp. Jt Dis.*, Oct. 2, cited by KLEINBERG.
WILENSKY, A. O. (1938), *Arch. Surg.*, **37**, 371.
WHEELER, K. W. (1941), *J. Urol.*, **45**, 467.

RAZOR BLADE IN ŒSOPHAGUS: TRANSTHORACIC REMOVAL

BY T. HOLMES SELLORS, LONDON

THE majority of foreign bodies that are swallowed by children produce little ill effect, but occasionally an awkwardly shaped object becomes lodged in the œsophagus. Endoscopic removal of the latter is successful in most instances and more drastic surgical measures are thereby avoided. When, however, the foreign body is so placed that it cannot be dealt with by œsophagoscopy, recourse to open operation may have to be considered. The fear of a fatal mediastinitis is present in every case of œsophageal ulceration and perforation and has relegated œsophagotomy into the category of dangerous operations.

The swallowing of a safety-razor blade by a small child is the subject of this case report. The blade became lodged in the œsophagus at the level of the aortic arch without producing many symptoms. Its discovery was more or less accidental and endoscopy showed it deeply embedded in œsophageal mucosa and unsuitable for removal by that method. Removal was effected by a transpleural approach to the œsophagus, which was incised. The blade was removed and the œsophagus sutured. Pleural infection developed, but this was treated and complete recovery resulted.

CASE REPORT

Male child, born September, 1942.

First attended Queen Elizabeth Hospital for Children under the care of Dr. Helen Mackay in March, 1945, with a history of occasional pyrexia and no gain in weight for about ten months. During the last six months he had frequently swallowed "buttons, small balls, and wheels from toys", etc.

In October, 1945, the boy was again brought to outpatients with a vague history of feverish attacks and abdominal discomfort. Further interrogation showed that he had had some difficulty in swallowing solids.

He was admitted to hospital on Oct. 23, where the following investigations were made: Urine normal; W.B.C.'s 25,600 (polymorphonuclears 50 per cent;

lymphocytes 40 per cent); rectal swab *B. aertrycke* isolated. The child was feverish on one occasion, but was on an ordinary diet.

Routine X-ray examination on Oct. 25, showed razor blade in the œsophagus (*Figs.* 412, 413). On further questioning, the father gave a history of the boy's inability to swallow fluids twelve days previously. In hospital the ward sister stated that he ate an ordinary dinner slowly with no evidence of pain.

Oct. 25, *Œsophagoscopy* by Mr. Ben Cohen. (Anæsthesia: intratracheal gas–oxygen–ether.) At mid-thoracic level the lumen was occluded by swollen congested mucosa. With the application of cocaine 10 per cent, and adrenaline 1–1000, the upper end of the blade could be determined lying posteriorly and fairly well wedged in. A little pus and a few tags of macerated paper were removed. It was impossible sufficiently to shrink the mucosa to obtain a more adequate view of the rest of the blade.

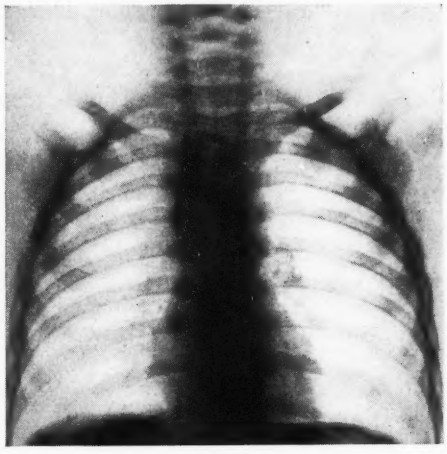


FIG. 412.—Anteroposterior radiograph of chest, showing razor blade in œsophagus.

FIG. 413.—Lateral view of chest.

Under the circumstances manœuvres such as removal in two parts after bisection along its length or removal after fragmentation, were abandoned as unpractical procedures.

The child was transferred to the Thoracic Surgical Unit at Harefield on Oct. 26. Œsophagotomy was the only method for removal and a transpleural approach was decided on. Extrapleural methods of approach in a child of this size were considered impracticable.

OPERATION (Oct. 26).—Anæsthesia (Dr. Parry Brown): intratracheal gas–oxygen and cyclopropane. Right thoracotomy with posterolateral removal of 4th rib. The œsophagus was identified and a mass felt in it at the level of, and just above, the azygos vein. The œsophageal wall was indurated and œdematous and the surrounding mediastinal structures were adherent. There was, however, no pus. The œsophagus was incised longitudinally and the razor blade withdrawn. The eyeholes and angled edges were entangled in mucosa, through which the foreign body had heavily ulcerated. The area was dusted with penicillin-sulphathiazole powder and the incision closed with two layers of fine thread (four interrupted stitches in each). The mediastinal pleura was left open and after further application of penicillin the chest was closed without drainage in the ordinary manner with the lung re-expanded.

PROGRESS.—The early convalescence was satisfactory and liquids and later soft solids were administered freely. Systemic penicillin, 2,435,000 units, was given over 25 days, as well as penicillin lozenges to suck, but about the seventeenth day fever and signs of toxæmia became apparent. Routine aspiration of the chest revealed no pus and this was attempted over the course of 12 days on fourteen occasions. Finally, it was decided to explore the chest deliberately to exclude the possibility of a para-œsophageal abscess.

Under anæsthesia two further aspiration attempts were made with negative results. A small length of rib was resected and an extensive empyema opened. The pus was extremely viscid and mucoid, as would be found in cases in which the empyema communicated with a mucous surface (such as a bronchus). This accounted for the failure of aspiration, though wide-bore needles were used in the later attempts. Ordinary drainage was established and the lung steadily re-expanded with complete healing 18 weeks after the original operation. At no time was there any evidence of free communication between œsophagus and pleura as judged by swallowed food and dyes, though the character of the pus indicated that primary healing of the œsophageal wall had not occurred.

The child has remained perfectly fit and has gained weight. There has been no difficulty in swallowing since the operation, though the remote possibility of a later stricture must be borne in mind.

Comment.—The razor blade when removed was partly covered in wrapping paper which may have prevented the condition from being worse than it was and have saved the mouth and pharynx from laceration when swallowing this object. Most of the paper had been dissolved by the time of operation and it was clear that œsophagotomy was the only possible method of removal. Any attempt to shift or break up the razor blade through the œsophagoscope might easily have resulted in incision of the aortic arch or azygos vein; the possibility of this was only too obvious at operation.

DISCUSSION

The case history is given in some detail since the discovery of the razor blade was 'accidental' in that its presence was only recognized by routine radiography; otherwise the presence of *B. aertrycke* in the stools might have been regarded as adequate reason for the fever and malaise which was the original cause of his attendance. Transpleural œsophagotomy is a method of dealing with impacted foreign bodies in the thoracic œsophagus and is liable to severe complications, but with the use of penicillin and adequate drainage of the mediastinal tissues (into the pleural cavity) infection should be capable of control.

CHORIONCARCINOMA OF THE TESTIS WITH GYNÆCOMASTIA

REPORT OF A CASE WITH EARLY BREAST CANCER

By ALEXANDER LYALL, Greenock

Gynæcomastia, or abnormal enlargement of the male breast, may be found in association with testicular tumours, atrophy of the testis, and certain tumours of the adrenal cortex. There is also a group of cases where no apparent cause for the enlargement can be found.

The following is the report of a case which was associated with a chorionepithelioma of the testis.

CASE REPORT

History.—G. B., aged 24, soldier, was admitted to hospital from overseas with the history that he had had his left testis removed two and a half months previously for a tumour. He had made a good recovery from the actual operation, but for a few weeks previous to his admission he had been losing weight and had noticed that his breasts were becoming tight and swollen.

Fig. 414.—Kidney with secondary chorioncarcinoma.

On Examination.—There was evidence of recent loss of weight. Both breasts were enlarged to about the size of the normal average virginal breast. The breast substance had a firm, slightly nodular consistence and a small amount of clear fluid was expressed from the nipple on pressure. The organs were slightly tender on palpation and the nipples and areolæ showed brown pigmentation. The left testis was absent and the scar was soundly healed. An X-ray examination of the chest showed the presence of multiple, small, secondary tumours. A Zondek-Aschheim test was carried out with the patient's urine and gave a strongly positive reaction.

The condition of the patient rapidly deteriorated, secondary tumours appeared in the abdomen, and he died five weeks after admission.

Post-mortem Examination.—The body showed advanced emaciation. Both lungs were riddled with secondary tumours, most of them being small, but the largest reaching a diameter of 4 cm. Necrosis and massive hæmorrhage were present in the larger ones, and in the basal region of the right lung there was a soft mass the size of a large orange which was composed of blood-clot with a nodule of white tumour at one side. The mediastinal glands were massively involved with tumour, forming a large mass pushing the heart forward. Multiple small secondaries were present in the liver, many of them showing the usual necrosis and hæmorrhage. In the right kidney there was a nodule measuring 3 cm. in diameter and bulging from the surface of the organ under the capsule. Much hæmorrhage had occurred into it (*Fig.* 414). Secondary deposits of tumour were found in the para-aortic glands. Section into the breast tissue showed it to be firm and nodular and to have a diffuse white colour. No obvious cystic changes were apparent. There was no fatty tissue present. Permission to examine the head was withheld, so that the pituitary gland was not examined.

Histology.—Tumours of the lungs, liver, kidney, and para-aortic glands were sectioned and examined by various staining methods. Various sections from both breasts were also examined. The tumour had the typical histology of chorioncarcinoma. The Langhans cells were cuboidal or polyhedral in shape, with clear cytoplasm which took on a slightly basophilic hue, and with nuclei which were vesicular but hyperchromatic in places. The syncytial elements consisted of irregular masses of acidophilic protoplasm containing oval nuclei of various sizes, with a dense irregular chromatin network. The two constituents were intimately mingled in many areas (*Figs.* 415, 416). Many areas of necrosis and hæmorrhage were seen, this being particularly marked in the lungs.

Sections of the breast tissue showed that the organ was packed with ducts and acini separated by fibrous stroma. The ducts were lined with columnar epithelium, the nuclei of which were elongated and colloid. Most of the ducts were dilated to some extent and contained an acidophilic, homogeneous material in which colostrum cells and fat globules were apparent (*Fig.* 417). Around each duct and acinus there was an "accommodative space" of loose fibrous stroma, the connective tissue outside this being dense and darkly stained. At places anaplastic changes were noted in the epithelium and there was a tendency towards spraying out of the cells into the surrounding stroma. At one point this anaplasia had advanced to the stage of definite malignancy. The ductule and acinus were infiltrated and replaced by a solid mass of cells which showed much variation both in size and shape (*Fig.* 418). The nuclei tended to be oval and colloid, with irregular chromatin content, and mitotic figures were abundant, representing every stage of cell division (*Fig.* 419). Giant cells with irregular, dark nuclei were seen at places (*Fig.* 420). The tumour bore no histological resemblance to the chorioncarcinoma, and had obviously arisen from breast tissue.

DISCUSSION

This case raises two interesting problems:—

1. Cause of the Gynæcomastia.—Gynæcomastia in testicular tumours has been noted by Heidrich et al. (1930), Le Sas (1931), Bergeret et al. (1937), Entwistle and Hepp (1935), and Gordon-Taylor and Till (1938). Kriss (1930, 1931) pointed out that the condition arose in only 5·7 per cent of all cases of testicular chorioncarcinoma. Moreover, there is still doubt as to why it should occur at all, Gordon-Taylor (1938) adding, "Opinion is not yet finally settled as to the hormonal origin of these changes, Bruno Kriss affirming that if a

hormonal cause were solely responsible, a higher percentage of chorionepithelioma of the testicle would show it."

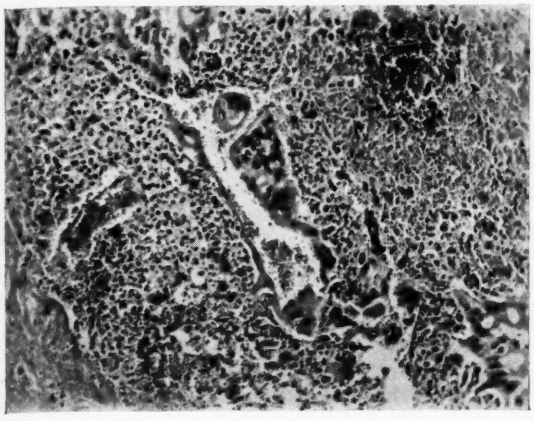


FIG. 415.—Low-power microphotograph showing the two component tissues of the chorioncarcinoma. Areas of necrosis are also seen.

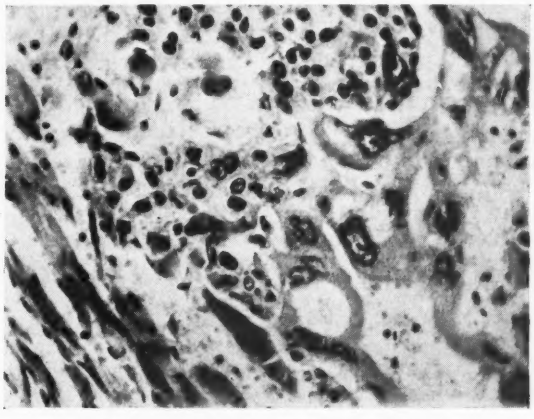


FIG. 416.—High-power view of chorioncarcinoma showing the mingled syncytium and Langhans cells.

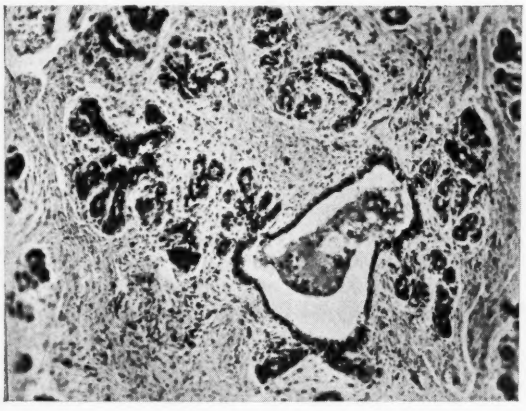


FIG. 417.—Low-power view of breast, showing a dilated duct containing homogeneous secretion and colostrum corpuscles. Many acini are also seen.

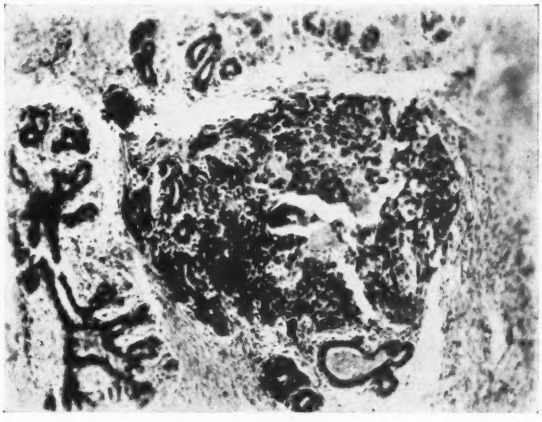


FIG. 418.—Low-power microphotograph, showing a mass of carcinomatous tissue growing from a duct.

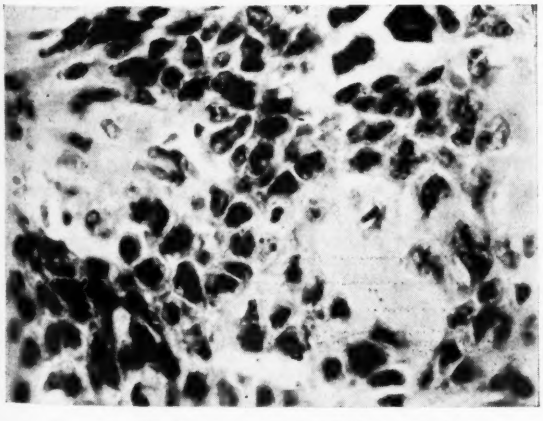


FIG. 419.—High-power section of breast tumour. Many mitotic figures are seen.

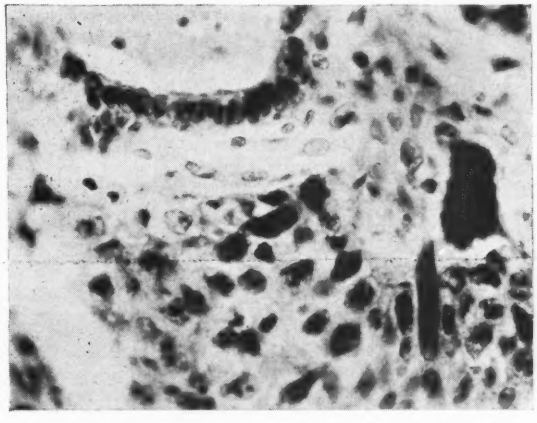


FIG. 420.—High-power view of breast tumour, showing large hyperchromatic nuclei.

In considering the problem it is natural to look to the parent tissue of the tumour for the responsible hormone. According to Novak (1941) the placenta produces three hormones. The best known is the one which forms the basis of the Zondek-Aschheim test, the gonadotrophic or anterior pituitary-like hormone. As this hormone is excreted in large amounts in many cases of testicular teratomata which show no evidence of breast changes, it is unlikely to be the one for which we are searching.

Progesterone and œstrogen are also produced in the placenta during certain stages of pregnancy. The effect of prolonged injections of œstrogenic substances in experimental animals is well known. Lacassagne (1932, 1936, 1938) did his work on mice, Geschikter et al. (1934) worked on monkeys, and Burrows (1935) on mice. The hyperplasia in these experiments appeared to have involved mainly the ductal system, suggesting that another factor is necessary to give the associated acinar changes of true gynæcomastia. The physiologists have suggested that whereas œstrogen plays the important role in building up the ductal system, it is the progesterone which develops the secretory acinar portion. That the pituitary may play an important part by means of some intermediate secretion is suggested by the work of Gomez and Turner (1937), who showed that neither œstrogen will stimulate breast growth in hypophysectomized rats. In their case of gynæcomastia Entwistle and Hepp (1935) noted a preponderance of the clear chromophobe cells usually associated with pregnancy. Bergeret et al. (1937) also described changes in the pituitary like those of pregnancy in their case.

2. Secondary Malignant Degeneration in the Breast.—Lacassagne (1938) succeeded in carrying the œstrogenic hyperplasia of the breasts in his male animal experiments on to the stage of frank carcinoma. In discussing their cases of stilbœstrol gynæcomastia in the human subject, Scarff and Smith (1942) mention the presence of dangerous epithelial hyperplasia in the ducts, but there was no actual malignancy. Our case would appear to be the first in which a true gynæcomastia in the human subject due to hormonal influence has progressed to the stage of malignant degeneration.

SUMMARY

A case of gynæcomastia due to secondary chorion-carcinoma is described. The most interesting feature of the case is the presence of early carcinoma in the hyperplastic breast.

REFERENCES

BERGERET, A., CAROLI, J., MILLOT, J. L., and SIMMONET, H. (1937), *Bull. Soc. méd. Hôp. Paris*, **53**, 849.
BURROWS, H. (1935), *Brit. J. Surg.*, **23**, 191.
ENTWISTLE, R. M., and HEPP, J. A. (1935), *J. Amer. med. Ass.*, **104**, 395.
GESCHICKTER, C. F., LEWIS, D., and HARTMAN, C. F. (1934), *Amer. J. Cancer*, **21**, 828.
GOMEZ, E. T., and TURNER, C. W. (1937), *Proc. Soc. exper. Biol.*, **37**, 607.
GORDON-TAYLOR, G., and TILL, A. S. (1938), *Brit. J. Urol.*, **10**, 1.
HEIDRICH, L., FELLS, E., and MATHIAS, E. (1930), *Beitr. klin. Chir.*, **150**, 349.
KRISS, B. (1930), *Arch. Gynäkol.*, **141**, 503.
— — (1931), *Ibid.*, **143**, 327.
LACASSAGNE, A. (1932), *C. R. Acad. Sci., Paris.*, **195**, 630.
— — (1936), *Amer. J. Cancer*, **27**, 217.
— — (1938), *C. R. Soc. Biol. Paris*, **129**, 641.
NOVAK, E. (1941), *Gynecological and Obstetrical Pathology*, 4. Philadelphia and London: W. B. Saunders Co.
SAS, L. (1931), *Brit. J. Urol.*, **3**, 31.
SCARFF, R. W., and SMITH, C. P. (1942), *Brit. J. Surg.*, **29**, 393.

THE ARNOLD-CHIARI MALFORMATION

By G. H. STEELE
SURGEON, ROYAL SURREY AND ST. LUKE'S HOSPITALS, GUILDFORD

IT is common knowledge that hydrocephalus is frequently encountered in association with, or following operation for, meningocele and meningomyelocele. What is not generally realized is that the malformation described originally by Arnold in 1894 and by Chiari a year later, which consists essentially of herniation of the hind-brain through the foramen magnum, is a common finding in these cases and that its recognition gives the clue to the correct line of treatment when the absence of associated congenital deformities offers a reasonable prospect of useful survival.

The development of thought with regard to these lesions is fascinating in the extreme. Morgagni (1769) advanced the thesis that the hydrocephalus, by reason of increased cerebrospinal pressure, was the primary cause of the meningeal bulge, but the frequent occurrence of meningocele without concomitant hydrocephalus did not fit in with this conception. Solovtzoff (1901) and Gredig and Schwalbe (1907), who christened the condition with its present name, followed Arnold and Chiari in publishing further anatomical observations. The nature of the malformation was more fully illustrated by the first British contribution to the subject, that of Russell and Donald (1935). They noted their findings in 10 post-mortem examinations and described a tongue of cerebellum and medulla, complete with choroid plexuses and enclosing an elongated fourth ventricle, protruding downwards through the foramen magnum and lying on the posterior surface of the spinal cord. The cervical nerve-roots pursued a course upwards instead of downwards to their foramina of exit, showing that the whole cord was displaced caudally; and a variety of associated abnormalities was encountered, such as hypoplasia of the medulla, non-differentiation of the cerebellum into vermis and lateral lobes, and closure of the aqueduct of Sylvius and foramina of Magendie and Luschka. They showed by injections of Indian ink that the cerebello-medullary hernia might obstruct the subarachnoid circulation of cerebrospinal fluid or block the fourth ventricle in addition, producing a communicating or non-communicating hydrocephalus accordingly; Dandy and Blackfan (1934) had already shown that three-quarters of the cerebrospinal fluid is absorbed in the skull and only one-quarter in the spinal canal. Penfield and Coburn (1938) suggested that the plug was a traction-cone due to the fact that the spinal cord became tethered in the region of the spina bifida and did not rise in the canal as the baby grew

and that the plugging effect was similar to that produced by the pressure-cone caused by a rise in intracranial pressure, the only difference being that in the first case the '*vis*' was '*a fronte*' and in the second '*a tergo*'.

Ingraham and Scott (1945), in a masterly series of articles, have recounted their experiences in a group of 462 patients with spina bifida, 98 of whom had meningoceles and 279 meningomyeloceles, the differentiation depending on whether nerve elements were or were not visible in the sac and on the presence or absence of neurological changes—weakness, sensory impairment, or sphincter disturbances. They recorded their findings in 20 autopsies, all exhibiting the Arnold-Chiari malformation, and their conclusions were similar to those of Russell and Donald, but in addition they described the co-existence of plastic meningeal exudate, microgyria (small 'wormy' cerebral gyri, more numerous than normal) and craniolacuna or *Luckenschadel* (diffuse rounded defects in the skull separated by bony ridges). The latter condition has been described by Vogt and Wyatt (1941) as frequently demonstrable radiologically in conjunction with meningomyelocele and meningocele, particularly the former. Microgyria and craniolacuna occurred in a high percentage of Ingraham and Scott's cases, but this is not surprising considering that most of them had meningomyeloceles. Jefferson (1942) operated on an adult patient with symptoms of high cervical tumour and discovered an Arnold-Chiari malformation; he then had the spine X-rayed and found a sacral spina bifida. This probably accounts for the cases reported by Aring (1938) and McConnell and Parker (1938), where the Arnold-Chiari malformation alone was found, but no mention was made of excluding the coexistence of spina bifida occulta.

The practical application of these considerations, which concerns primarily the general practitioner, the obstetrician, and the general surgeon, is the question, "Is it worth while operating on a meningocele?" There are probably no two opinions on the unwisdom of operating on a meningomyelocele, and the selection of cases is obviously limited to those unassociated with irremediable deformities or gross neurological changes (in a very young infant the latter boils down to whether it kicks its legs properly, its skin is relatively insensitive, and it has not yet developed a social conscience with regard to its sphincters), and the absence of coexisting hydrocephalus. It would appear reasonable to suppose that the cord is less likely to be tethered by a meningocele than by a meningomyelocele; and of Ingraham and Scott's 462 cases 60, or 13 per cent, are now considered normal and another 13 per cent are suffering from mild neurological disability. And if such a reasonable percentage of affected children will lead useful lives, and if of those who develop hydrocephalus a large proportion manifest the Arnold-Chiari malformation which is amenable to surgery, more of these cases will be considered to be worth operating on.

The operative procedure is obvious—decompression of the herniated hind-brain by removal of the posterior part of the bony canal in which it is incarcerated; this is a very simple proceeding, and according to Jefferson can be performed at almost any age. The operation is performed through a vertical midline incision; the lower part of the occipital bone, the posterior part of the foramen magnum, and the posterior arches of the cervical vertebræ are removed to below the level of the lower edge of the medulla and the dura is widely opened. The muscles and skin are closed in layers in the usual fashion.

When considering the age at which operation should be undertaken it must be borne in mind that some meningoceles are obviously in danger of immediate rupture of their thin covering membrane

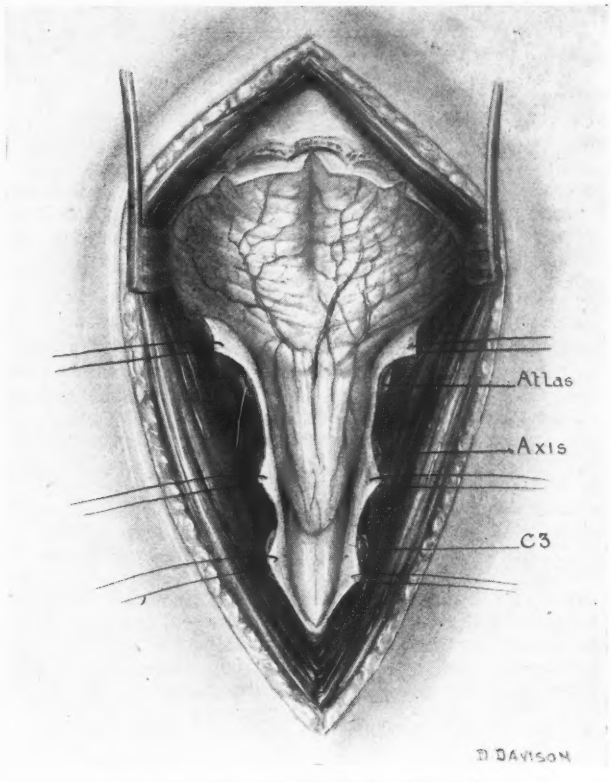


FIG. 421.—The Arnold-Chiari malformation.

and will have to be operated on shortly after birth; but if there is no urgency of this sort it would appear that the choice lies between operating on the meningocele as soon as is reasonable and hoping that hydrocephalus will not develop, or performing decompression if, and as soon as, it does (by which time the brain may already have suffered irreparable damage), or doing a routine decompression soon after the cure of the meningocele on the grounds that a high percentage of cases are associated with the Arnold-Chiari malformation. Ingraham's opinion is that the majority should be left till the age of 12 to 18 months to permit better assessment of neurological disabilities and hydrocephalus, and that the decompression should be performed 10–14 days after the meningocele operation.

The following case illustrates many of the above points.

A female child was born with a meningocele, the covering of which was so thin as to be liable to immediate rupture. It moved its legs well, and the parents, who

were fully informed of the likelihood of associated abnormalities, were most anxious to have the child's life preserved. The meningocele was therefore excised without anæsthesia when the baby was 5 hours old; no neural elements were observed in the wall of the sac, but the gap was so large that skin-flaps extending on to the buttocks and up over the shoulders had to be utilized to cover it. A cerebrospinal fistula appeared at the junction of the flaps, but dried up in a fortnight, when the child was discharged in perfect health. Four months later it began to show signs of hydrocephalus; the circumference of the head increased from 18¾ in. at the age of 7 months to 19¾ in. at 13 months and 20¼ in. at 16 months, the corresponding average measurements in normal girl children being 17 in., 17¾ in., and 18 in, with a variation of half an inch either way (Tredgold, 1937). The child appeared listless and took no interest in its surroundings. It was then sent by its doctor to a neurological hospital whence it returned with an offer to cauterize its choroid plexuses; this was refused. In the meantime I had been informed (Jefferson) of the existence of the Arnold-Chiari malformation; I thereupon performed a cerebello-medullary decompression and found a perfect example of the condition (*Fig.* 421). The bone, being soft, was very easy to remove, and on opening the dura the herniated cerebellum and flattened medulla were seen to extend on the posterior surface of the cord as far downwards as the posterior arch of the third cervical vertebra, which was included in the decompression. No exudate or adhesions were seen in the subarachnoid space; it was not, of course, possible to ascertain the condition of the cerebral gyri, but there was no radiological evidence of craniolacuna. Suture of the upper part of the wound in layers was difficult on account of the absence of layers and cerebrospinal fistula appeared here. The child became drowsy and irritable a week after operation and its condition gave rise to considerable anxiety, but the fistula closed spontaneously and the child was again discharged.

The head measurements have reverted to a normal rate of increase, and the parents are delighted. They say that the child at 6 months after operation is much brighter and more interested in its surroundings and is beginning to talk, and clinical observation confirms this favourable impression. It will be some considerable time, however, before it is possible to assess the extent of the cerebral recovery; my only regret is that I was not aware earlier of the existence of the syndrome, so that the development of hydrocephalus could have been arrested or prevented before the brain became distended. The child has in addition a mild degree of congenital dislocation of one hip which hinders its attempts at walking, but which should respond readily to treatment, and slight impairment of circulation in the same leg, which is already improving spontaneously.

Comment.—The advice given to many parents in the past has been that in view of the gloomy outlook it would be advisable to give up and start another pregnancy, or if the meningocele has been operated upon the end-results have often been deplorable; but with a judicious selection of cases there appears to be no reason for the continuation of an attitude of undue despondency.

I am most indebted to Professor Jefferson for educating me in this particular aspect of neurosurgery, and for his continued guidance in the management of the case.

REFERENCES

ARING, C. D. (1938), *J. Neurol. Psychiat.*, **1**, 100.
ARNOLD, J. (1894), *Beitr. path. Anat.*, **16**, 105.
CHIARI, H. (1895), *Denkschr. Akad. Wiss. Wien*, **63**, 71.
DANDY, W. E., and BLACKFAN, K. D. (1934), *Amer. J. Dis. Child.*, **8**, 406.
GREDIG, M., and SCHWALBE, E. (1907), *Beitr. path. Anat.*, **40**, 132.
INGRAHAM, F. D., and SCOTT, H. W. (1945), *New. Engl. J. Med.*, **228**, 559 et seq.
JEFFERSON, G. (1942), personal communication.
MCCONNELL, A. A., and PARKER, H. L. (1938), **61**, 415.
MORGAGNI, G. B. (1769), *The Seats and Causes of Disease*, **1**, 12, 244.
PENFIELD, W., and COBURN, D. F. (1938), *Arch. Neurol. Psychiat.*, **40**, 328.
RUSSELL, D. S., and DONALD, C. (1935), *Brain*, **58**, 203.
SOLOVTZOFF, N. (1901), *Nouv. Iconogr. Salpêt.*, **14**, 118.
TREDGOLD, A. F. (1937), *Textbook of Mental Deficiency*, 530.
VOGT, E. C., and WYATT, G. M. (1941), *Radiology*, **36**, 147.

THE TRANSFORMATION OF AN EMBRYONIC LIPOMA TO A COMMON LIPOMA

BY D. P. VAN MEURS
ASSISTANT SURGEON COUNTY HOSPITAL, FARNBOROUGH

ALTERATION of the histology of a tumour is usually either towards a more malignant form or of a degenerative nature. The tumour described here adopted the opposite course, and finally an innocent tumour was removed with every hope of a permanent cure, when the earlier histology had suggested that the prognosis might be unfavourable.

The case history and pathology are described, and there is a brief discussion on the site of origin of the tumour and the reasons for its transformation.

CASE REPORT

HISTORY.—The patient, F. C., female, was first seen as an out-patient on Feb. 19, 1943, when she was five months old. The history was of a swelling in the right axilla, which had been first noticed by an intelligent mother two days previously, slight swelling of the right arm, and prominence of the right pectoral veins. No pain seemed to be present, but the mother noticed that the baby seemed to use the right arm less than the left. Otherwise the baby was perfectly well. The baby was well developed and healthy. The right arm showed slight non-pitting œdema from the axilla to the wrist, and the pectoral veins were prominent. There was a moderately tense, round swelling visible in the axilla, over which normal skin moved freely. There was no translucency and only doubtful fluctuation in the tumour, and no swelling could be felt in the posterior triangle of the neck. Radiography of the chest and axillary region revealed no abnormality, and the baby was admitted to hospital next day without a definite diagnosis being made.

FIRST OPERATION, Feb. 23 (patient aged five months).—Aspiration of the tumour produced no fluid. Through

an axillary incision the tumour was dissected out with some difficulty. It was well encapsulated but extended behind the axillary contents as far as the vertebral border of the scapula. The general appearance was that of a lipoma. It was lobulated, yellowish in colour, the centre of each lobule having a pinkish hue.

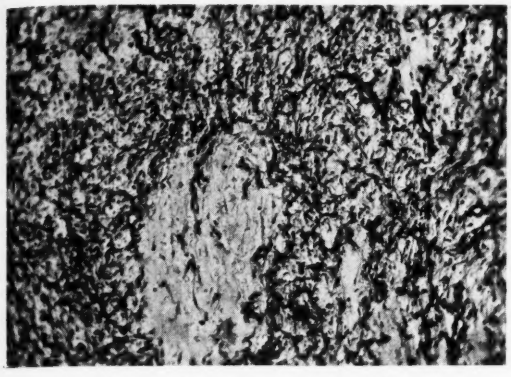


FIG. 422.—Microscopical appearance of section of tumour removed at five months. Hæmatoxylin and eosin. (× 75.)

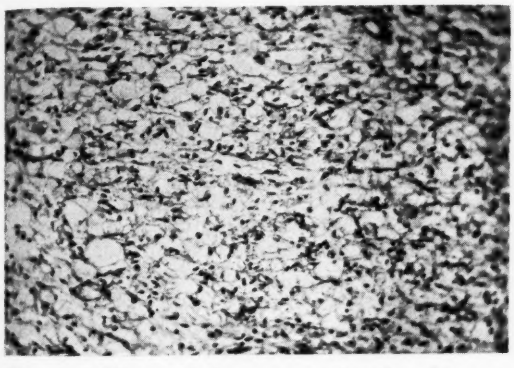


FIG. 424.—Microscopical appearance of section of tumour removed at eleven months. H. and E. (× 75.)

The patient was discharged from hospital in twelve days and then attended as an out-patient. Two months later a similar swelling was present in the posterior triangle of the neck and the patient was readmitted.

SECOND OPERATION, April 28 (patient aged seven months).—The tumour was dissected out through an incision placed over it. It was encapsulated, except deeply, where it was probably adherent to the site of the previous operation.

A further recurrence was noted in the same position four months later.

THIRD OPERATION, Sept. 5 (patient aged eleven months).—The tumour was again removed through the posterior triangle of the neck.

Two months later a swelling was apparent in the axilla, and with bimanual examination it could also be felt in the posterior triangle.

FOURTH OPERATION, Nov. 15 (patient aged thirteen months).—The tumour was removed through combined incisions in the axilla and the posterior triangle.

No further recurrence was observed until seven months later, and it was decided to postpone further operation until the patient was older.

FIFTH OPERATION, Oct. 2, 1944 (patient aged twenty-five months).—The tumour was visible in both the axilla and posterior triangle. An incision was made above and parallel to the inner two-thirds of the clavicle, the medial end of the incision then turning downwards and outwards to the anterior fold of the axilla. The pectoral muscles and the clavicle were divided. The tumour lay behind the main vessels and nerves, but sent prolongations between the nerve-trunks. It was adherent to the scalene muscles and serratus magnus and to the long thoracic nerve, and was removed with some difficulty. No thrombosis was present in the main veins.

The patient was discharged from hospital four weeks later with the clavicle apparently united, but with a

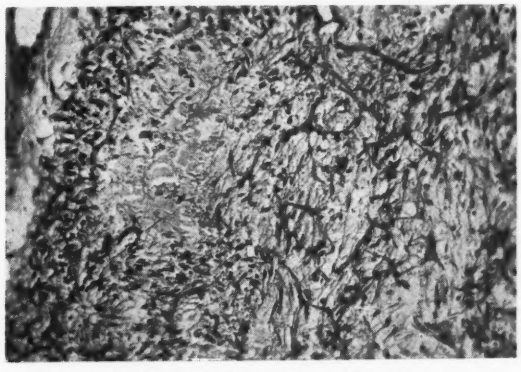


FIG. 423.—Microscopical appearance of section of tumour removed at seven months. H. and E. (× 75.)

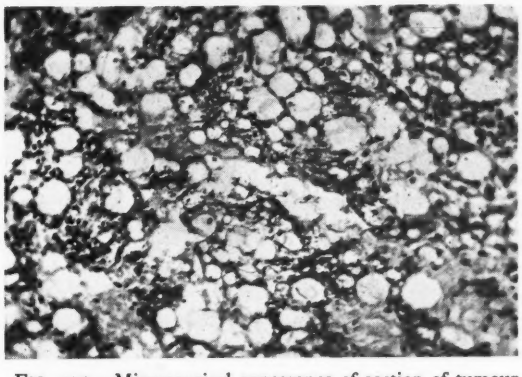


FIG. 425.—Microscopical appearance of section of tumour removed at thirteen months. H. and E. (× 75.)

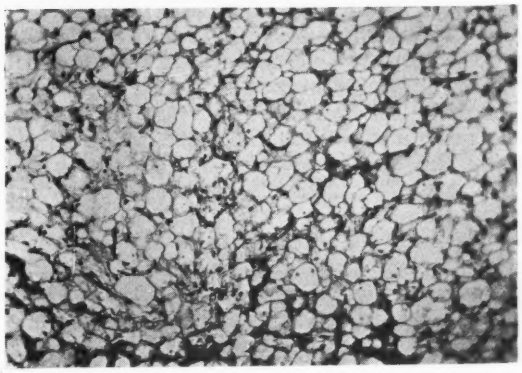


FIG. 426.—Microscopical appearance of section of tumour removed at twenty-five months. H. and E. (× 75.)

paralysis of the long thoracic nerve. Six months later, after conservative methods had failed, she was readmitted for repair of the ununited clavicle. The tumour had not recurred up to eleven months after the last operation.

After each operation the swelling of the arm diminished and the pectoral veins became less prominent, but these conditions recurred with the tumour. The patient's health and development remained excellent, and repeated radiographs of the chest showed no abnormality.

HISTOLOGICAL APPEARANCES (Prof. G. Payling Wright) (*Figs.* 422–426).—Paraffin embedded sections from specimens fixed in formol-saline were stained with

hæmatoxylin and eosin, with Van Gieson's stain for connective tissue, and by Mayer's muci-carmine method. Frozen sections, stained with Sudan III, were prepared to determine the amount and distribution of the fat.

Materials taken from all five resected masses had fundamentally the same basic histological structure. They were all encapsulated with a thin layer of fibrous tissue, and were rather more extensively vascularized than an ordinary lipoma. Much the greater part of the masses, especially the earlier ones, was composed of a loose, irregularly arranged, network of fine spindle cells with elongated, rather deeply staining, nuclei. These cells tended to be more numerous and more densely packed immediately beneath the capsule and in the vicinity of the main connective-tissue septa. The spaces between the cells were filled with a finely reticular material which stained pinkish with the muci-carmine stain. The appearance generally was closely similar to that seen in a myxoma. There was little evidence of growth, and in an extensive search no mitotic figures were found.

In the frozen sections, some of the characteristic cells were seen to be swollen in varying degrees and to contain fat: in some it was present in finely divided droplets, but in the majority of such cells it could be seen as a single large drop resembling that seen in a fully developed adipose tissue cell. Heavily fat-containing cells of this latter kind were often aggregated into small lobular masses, so that areas of a frankly lipomatous nature were interspersed with others that more closely resembled a myxoma.

The main difference observed in the material removed at the successive operations was the progressively increasing proportion of tissue of ordinary lipomatous appearance. In the earlier specimens, the myxomatous areas tended to preponderate, but in the last specimen, removed twenty months after the first one, the whole appearance was scarcely distinguishable from that of a common lipoma.

DISCUSSION

The recurrences after operation were doubtless in part due to incomplete removal. This was realized at the time, but I was unwilling to inflict an extensive operation on an infant or to face becoming involved in the important structures in a small axilla. Obviously something had to be done from time to time to relieve the pressure on the axillary vein, and the operations performed seemed to achieve this object. The gradual alteration in the histology of the tumour was thought to justify this decision, although the position was rather difficult after the first recurrence. The sections were then seen by several histologists and malignancy was not excluded. No reference could be found to the occurrence of liposarcoma or embryonic lipoma in an infant, and the view was taken that the embryonic appearance was due to the age of the patient and the site of the tumour.

Site of Origin.—Hatai (1902), Bonnot (1908), and Shattock (1909) each described adipose tissue in the axilla and root of the neck in which the fat cells could be differentiated from those found in ordinary adipose tissue. The fat in the cells tends to be in the form of fine droplets. There are also spindle-shaped cells resembling fibroblasts, lymphoid tissue, and the whole structure is fairly vascular. Even in a five-months-old fœtus the surrounding adipose tissue is of the adult type, and this specialized structure can be differentiated from adipose tissue in adult life (Shattock). Hatai first suggested that this tissue represents the hibernating gland of animals, best developed in the hedgehog and dormouse. Whether this organ is different from the so-called dark adipose tissue present in the prevertebral and inguinal regions is uncertain, but there is a great histological similarity. However, in all these situations adipose tissue of an embryonic type tends to persist, and it is of interest that liposarcomata tend to occur in the scapular and prevertebral regions. There is also a possible tendency for common lipomata to appear in those regions where normal deposits of fat occur in the animal world or as human racial peculiarities. The camel's hump, the fatty tails of marsupials and certain breeds of sheep, the fat thighs of the ancient Egyptian women, and the fat buttocks of Bush-women, all have their counterparts in lipomatous tumours.

It is probable that this tumour arose from the persistent embryonic adipose tissue in the interscapular gland.

Histological Changes.—The endocrine control over fatty deposits is evident in pituitary disease and cretinism. The occurrence of multiple lipomata, often symmetrical, suggests that lipoma formation may also be the result of failure or perversion of a general controlling influence, possible endocrine. The existence of mixed lipomata (myolipomata, fibrolipomata) and lipomata connected with spina bifida suggests that the controlling factor is a general one affecting the development of many types of connective tissue from a common embryonic cell.

A generalized fatty deposit may be a reversible state, but a lipoma seems to be as autonomous as any other tumour. Admittedly, as Shattock pointed out, a few lipomata have been described which may have diminished in size during emaciation, but there is some doubt as to the authenticity of these cases. Tumours, even if malignant, may be affected by endocrine factors, as shown by the effect of œstrin on some neoplasms. However, once a neoplasm has formed, it rarely alters except to a more embryonic or malignant type, although it is possible that the histology and rate of growth are influenced by a general controlling factor.

The tumour under discussion appears to have arisen from persistent embryonic tissue at a time of life when the transformation from connective tissue to fat is very active (Geschickter, 1934). The factors controlling cell growth at this stage of development must be very strong, and, although originally embryonic, the tumour seems to have remained in touch with these factors sufficiently to be altered into at least an obviously innocent tumour.

SUMMARY

1. Serial sections are shown of the transformation of an embryonic lipoma to an adult-celled lipoma.

2. The site of origin of the tumour and the reasons for its transformation are briefly discussed.

My thanks are due to Professor G. Payling Wright for the pathological report and for his helpful criticism of this article. I should also like to thank Mr. J. E. Andrews, of the Southern Group Laboratory, for taking the microphotographs.

REFERENCES

BONNOT, M. (1908), *Anat. and Phys.*, **43**, 43.
GESHICKTER, C. F. (1934), *Amer. J. Cancer*, **21**, 617.
HATAI, S. (1902), *Anat. Anz.*, **21**, 369.
SHATTOCK, S. G. (1909), *Proc. R. Soc. Med.*, **2** (Path. Sect.), 252.

MASSIVE HÆMORRHAGE FROM A PEPTIC ULCER IN A MECKEL'S DIVERTICULUM IN A FEMALE CHILD OF ELEVEN MONTHS

By R. W. WILKINSON

LATE HOUSE PHYSICIAN, CHILDREN'S DEPARTMENT, GENERAL INFIRMARY AT LEEDS

THIS case is of interest as it is one of the few on record where a successful operation was performed on a child below the age of one year for the relief of hæmorrhage from a peptic ulcer in a Meckel's diverticulum.

CASE REPORT

HISTORY.—M. D., a female child aged 11 months, was first admitted to another hospital on May 28 with melæna of one day's duration. The condition cleared up while in hospital, and the child was discharged. Bleeding recurred almost immediately, necessitating readmission on June 4. The stools varied in colour from dark brown to bright red, and the blood appeared to be intimately mixed with the fæces. On June 7 the hæmoglobin was 36 per cent and two transfusions totalling 180 c.c. were given into the bone-marrow. The bleeding continued intermittently, and as the temperature ranged between 99° and 102° F. a course of sulphasuxidine was begun on June 10. The hæmorrhage now ceased. Microscopical examination of the fæces on June 11 showed some mucus, but no blood, pus, or protozoa, and culture for organisms of the dysentery group was negative. On June 14 the bleeding time was 3 minutes, the clotting time 5 minutes, the platelet count 205,000, and the fragility and size of the red cells were normal. The child appeared to be losing ground, and as further bleeding occurred on June 28 she was transferred to the Leeds General Infirmary under the care of Professor C. W. Vining.

ON ADMISSION.—The child was well nourished, but pale and irritable. Full clinical and radiological examinations were negative. One large melæna stool was passed shortly after examination was completed. A blood-count showed red cells 1,500,000, hæmoglobin 28 per cent. Following transfusion (one pint Group O blood) there was oozing of fresh blood from the rectum. During the night of July 5 the child experienced a massive intestinal hæmorrhage of almost pure blood. On July 6 she received a further pint of Group O Rh + blood, after which her hæmoglobin rose to 78 per cent.

On the same day, Professor P. J. Moir was asked to see the child, and a provisional diagnosis of ulcerated Meckel's diverticulum or intestinal hæmangioma was made, exploratory laparotomy being suggested.

AT OPERATION.—Laparotomy was carried out on July 9. About 18 in. from the ileocæcal valve on the antimesenteric border of the small intestine a small rubbery white nodule was found. The tumour was removed, together with a portion of the surrounding bowel. Except for a little vomiting on the second day, convalescence was uneventful. The child was discharged on July 21 and no further bleeding has been noticed.

THE SPECIMEN.—For a report on the histology of the tumour, I am indebted to Dr. C. J. E. Wright, of the Pathological Department of the Infirmary: "Naked eye the specimen appeared to be a small elliptical portion of bowel wall with a white nodule measuring about 0·7 cm. diameter by 0·3 cm. in height projecting from the serosal surface. On section no evidence of tumour could be seen and the projection appeared to be a small Meckel's diverticulum.

"*Histology* (*Fig.* 427).—This is a Meckel's diverticulum showing a relatively large area of gastric heterotopia involving the wall of the Meckel together with the surrounding intestinal wall almost as far as one of the edges in the long diameter of the specimen. The gastric type mucosa shows typical oxyntic cells and the adjacent intestinal epithelium what is virtually a Peyer's patch. An ulcer is present in the diverticulum and

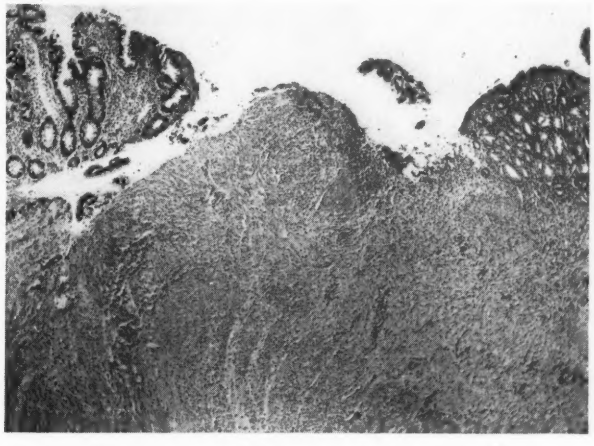


FIG. 427.—Microphotograph showing ulcer and gastric glands. (× 30.)

shows a small thrombosed vessel in the floor, quite possibly the source of the hæmorrhage. Owing to the presence of the heterotopic gastric mucous membrane the ulcer may be regarded as of peptic type."

DISCUSSION

Out of 30 diverticula examined by Schaetz (1925) 5 were found to contain gastric glands. Roughly the same incidence in heterotopia has been the finding of several other observers (Greenblatt, Pund, and Chaney, 1931; Curd, 1936). Brown and Pemberton (1936), however, discovered gastric mucosa in 13 cases out of 55 (24 per cent), Farr and Penke (1935) in 6 cases out of 13 (46 per cent), while Hudson and Koplik (1932) were able to demonstrate heterotopia in 67 per cent of a large series. If we regard the incidence as about 30 per cent, and the occurrence of Meckel's diverticulum as 2 per cent, then the possibility of gastric epithelium is roughly 1 in every 170 persons.

Deetz, in 1907, was the first to draw attention to the peptic nature of the ulceration, though cases had been reported by von Denecke (1902) and Hilgenreiner (1903). Guibal (1924) expressed the belief that it is this type of diverticulum which ulcerates, causes hæmorrhage, and occasionally perforates, with subsequent peritonitis. This is also borne out in the experience of Stulz and Worringer (1925), who state that the cause of the hæmorrhage or perforation is to be found in the ulceration of the heteroplastic gastric mucosa. That this is not the whole story, however, is shown by the fact that bleeding associated with heterotopia, but without ulceration, may occur (Abrams, 1938). With regard to this, Taylor (1927)

says: "A possible explanation is that an active acid secretion formed by the heterotopic glands may have devitalized the neighbouring intestinal epithelium and rendered it susceptible to an infection which would otherwise be easily overcome."

This condition is most frequent in children. Cobb (1936) found that 74 per cent of cases of peptic ulcer in a Meckel's diverticulum were in children from 1 to 15 years, while Matt and Timpone (1940) reviewing the literature, discovered that out of 76 histologically proved cases, 42, or just over 56 per cent, occurred under the age of 10 years, and 72, or roughly 94 per cent, were under the age of 30.

The cardinal sign is profuse repeated intestinal hæmorrhage. Pain is a very variable feature which may or may not be present. Should the ulcer perforate, there is superimposed the picture of an acute abdominal emergency.

The youngest case I have been able to find where a successful operation was performed is that of Fevre, Patel, and Lepart (1930), where a diagnosis of intussusception was made in a child aged 5 months who was bleeding from the rectum, and who had the signs of an acute abdomen: operation revealed a perforated Meckel's diverticulum which, histologically, showed an ulcer at the junction of the ileal and gastric mucosæ. Reports of similar cases under 1 year include those of Bunne (1936), Mason and Graham (1932), Moore (1926), Mallory (1936), Weiner and Seley (1939), Montgomery (1932) Conrad (1941), and Tisdall (1928).

The possibility of a diverticulum should be kept in mind in any case of profuse intestinal hæmorrhage, especially if repeated. In intussusception the blood is mixed with mucus, a tumour can generally be felt, either abdominally or more rarely (and fatally) per rectum, and there are the characteristic screaming attacks with drawing up of the legs. Local conditions such as rectal polypi or ulcerative colitis can be excluded by endoscopy. Bacteriological examination of the stools will show the presence of dysenteric organisms. Henoch's purpura may be ruled out by the absence of purpuric spots, of severe colic which is usually a feature, and of swelling and pain in the joints. Purpura hæmorrhagica shows a diminished platelet count. Hæmophilia is to be considered in the male child: here the disease may be manifest in the first six months of life (Price, 1941), but the family history and the blood findings will help to establish the diagnosis. Hereditary telangiectasia is so rare that mention is made only in passing. Rectal bleeding in the first few days of life is usually due to hæmorrhagic disease of the newborn or acute ulceration of the duodenum, but Moll's case (1926), where bleeding occurred intermittently from the second day up to the fifth month, and where post mortem a huge diverticulum with two chronic ulcers at its junction with the ileum was found, does suggest that peptic ulcer in a Meckel's diverticulum can also cause hæmorrhage at this early age. The most difficult conditions to exclude are an intestinal polyp or hæmangioma, but in both cases abdominal section will be required.

Treatment.—The treatment for the condition is removal of the diverticulum. Blood transfusion will be required in most cases as a pre-operative measure. Greenwald and Steiner (1931) give mortality figures of 7·6 per cent before perforation of the ulcer, and 58 per cent after perforation.

SUMMARY

A case of peptic ulcer in a Meckel's diverticulum in a female child aged 11 months is reported. Complete recovery followed removal of the diverticulum.

I should like to express my thanks to Professor C. W. Vining, Consulting Physician to the Children's Department, for his encouragement in writing this article; to Professor P. J. Moir, Professor of Surgery in the University of Leeds, for kind criticism and access to the operation notes; to Dr. L. Moss for notes on the child's progress before admission; and to the Sister and nurses of the Children's Department for their aid in the post-operative period.

REFERENCES

ABRAMS, A. L. (1938), *J. Pædiat.*, **13**, 513.
BROWN, P. W., and PEMBERTON, J. DE J. (1936), *Proc. Mayo Clin.*, **11**, 259.
BUNNE, F. (1936), *Z. Chir.*, **63**, 2362.
CHRISTIE, A. (1931), *Amer. J. Dis. Child.*, **42**, 544.
COBB, D. B. (1936), *Ann. Surg.*, **103**, 747.
CONRAD, H. A. (1941), *Amer. J. Surg.*, **52**, 267.
CURD, H. H. (1936), *Arch. Surg.*, **32**, 506.
DEETZ, E. (1907), cited by COBB.
VON DENECKE, K. (1902), *Ibid.*
FARR, C. E., and PENKE, M. (1935), *Ann. Surg.*, **101**, 1026.
FEVRE, M., PATEL, M., and LEPART (1930) *Bull. Soc. nat. Chir.*, **56**, 756.
GREENBLATT, R. B., PUND, E. R., and CHANEY, R. H. (1936), *Amer. J. Surg.*, **31**, 285.
GREENWALD, H. M., and STEINER, M. (1931), *Amer. J. Dis. Child.*, **42**, 1176.
GUIBAL, L. (1924), cited by CHRISTIE.
HILGENREINER, H. (1903), cited by COBB.
HUDSON, H. W., and KOPLIK, L. H. (1932), *New. Engl. J. Med.*, **206**, 827.
MALLORY, T. B. (1936), *Ibid.*, **214**, 481.
MASON, J. M., and GRAHAM, G. S. (1932), *Ann. Surg.*, **96**, 983.
MATT, J. G., and TIMPONE, P. J. (1940), *Amer. J. Surg.*, **47**, 612.
MOLL, H. H. (1926), *Brit. J. Surg.*, **14**, 176.
MOORE, C. U. (1926), in discussion on ABT, I. A., and STRAUSS, A. A. (1926), *J. Amer. med. Ass.*, **87**, 991.
MONTGOMERY, A. H. (1932), *Tr. West. Surg. Ass.*, **42**, 295.
SCHAETZ, G. (1925), cited by CHRISTIE.
STULZ, E., and WORRINGER, P. (1925), cited by CHRISTIE.
TAYLOR, A. L. (1927), *J. Path. Bact.*, **30**, 415.
TISDALL, F. F. (1928), *Amer. J. Dis. Child.*, **36**, 1218.
PRICE, F. W. (1941), *A Textbook of the Practice of Medicine.* London: Oxford Medical Publications.
WEINER, S. B., and SELEY, G. P. (1939), *J. Mt. Sinai Hosp.*, **5**, 620.

AMŒBIC GRANULOMA OF THE SKIN

By I. NORWICH
SURGEON-SUPERINTENDENT, CORONATION HOSPITAL*
AND D. A. MUSKAT
RESIDENT SURGICAL OFFICER, CORONATION HOSPITAL

AMŒBIC granuloma of the skin, or amœbiasis cutis, an uncommon lesion due to infection with the *Entamœba histolytica*, crops up periodically in the literature both on account of its rarity and the interesting pathological and clinical picture it presents.

E. histolytica may, under certain conditions, invade the skin and cause extensive ulcerative granulomatous lesions, but blood-stream invasion has never been reported. The lesions practically always arise by direct continuity from some other focus of the disease, e.g., drainage tract from the liver, the site of aspiration of the liver, around a colostomy wound, or from the rectum.

Although primary infection of the skin, according to Craig (1944), is reported rarely, a number of authors report ulcerative skin lesions around the anus. Crawford (1933) reports a case following an amœbic abscess of the perineum, the secondary lesion involving both buttocks. Engman and Meleney (1931) give an excellent description of the pathology of the skin lesions caused by the *E. histolytica*. According to these authors, it is characterized by a rapidly spreading ulcerative process, which, owing to the rapidity of its spread, has an irregular border and overhanging edge, from under which pus can be expressed, and a floor which is lined with indolent granulation tissue. The ulcer is painful and extremely tender.

Tixier and others (1927), under the name ' porodermie amibienne ', describe lesions of the peri-anal region. In their case (chronic dysentery of six years' duration) entamœbæ were found in the pus. Ngai and Frazier (1933) give a very complete review and describe 3 cases. L. F. Hamburger (1925) reported a case with a tumour in the right lumbar region, discharging liver pus. Sections of tissue from the edge of the ulcer showed, on suitable staining, the presence of amœbæ. F. L. and H. E. Meleney (1935) describe a very interesting case of a Chinese patient who, a month before admission, noticed a small, hard, tender lump to the right of the anus. This increased in size for three weeks, the patient being unable to sit down. Ten days prior to admission the lump burst, discharging foul pus. The patient was a severe diabetic, and on admission had a large, foul-smelling ulceration, extending from the upper margin of the sacrum to the upper third of the thigh and to the perineum. The line of demarcation was relatively smooth and sharply outlined. Four days after admission the ulcerative area was excised. Emetine hydrochloride was given as soon as the diagnosis was made, the diabetes being controlled at the same time. On the twenty-first day a Reverdin graft was applied, the patient subsequently being discharged, fully recovered.

Manson-Bahr (1938) describes a case of ulceration of the abdominal wall secondary to colostomy for supposed carcinoma of the rectum. *E. histolytica*, however, was found in sections of the ulcer, the condition clearing up on emetin therapy. W. B. Gabriel (1945) reports a case, very similar to that of the Meleneys, of extensive amœbic ulceration of the abdominal wall and buttocks. The primary lesion appeared to be a perirectal abscess. Treatment by emetine cleared up the infection. Thiersch grafts were subsequently applied, with complete healing. Manson-Bahr (1943) is of the opinion that amœbiasis cutis occurs chiefly as an unsuspected secondary infection of papillomata, usually of the anal region. More recently McConaghey (1945) states that only 19 cases of proved amœbiasis of the skin, perineum, and buttocks have been reported in the literature. Of these, the majority occurred in China. He describes a case of an Anglo-Burmese lady, who, after having blood and mucus in the stools, developed a blind peri-anal ' boil '. On admission she had a large fungating mass, the size of a walnut, around the anus. The serous exudate showed abundant trophic amœbæ. Here again, the condition cleared up completely with six doses of emetine.

In South Africa amœbic infection is of common occurrence, particularly in Natal, where the infection amongst the non-European community is quite high. On the Witwatersrand there is also quite an appreciable incidence, which Osburn (1943) attributes to two factors: (1) Exposure from the presence of natives from Natal and Nyasaland; and (2) Visiting of these territories by residents of the Witwatersrand.

From the above brief review it appears that amœbic infection of the skin of the buttocks is most unusual, and as such, any case presenting these features is worth recording.

The following case is therefore of considerable interest in that the pathological features of amœbic granuloma of the skin of the buttock are well demonstrated.

CASE REPORT

HISTORY.—I. v. St., a young adult male Griqua (coloured), aged 28 years, a painter by trade, was admitted on March 20, 1945, to the surgical wards of the Coronation Hospital, with the diagnosis carbuncle (?), sarcoma of the buttock (?). The following history was elicited: For the last year he had not been quite well, suffering from frequent attacks of colicky, lower abdominal pain, usually associated with diarrhœal stools. In the intervals, however, he was quite well, although he lost some weight during this period. There was no vomiting, no intermittent fever, malaise, headaches, or similar evidences of toxic disturbances. He occasionally noted some blood in the stools, but no mucus. Appetite during this period was good and there had been no general health disturbance. He had a slight cough for the last few years, accompanied by some sputum, but no hæmoptysis.

*A branch of the Johannesburg General Hospital, South Africa.

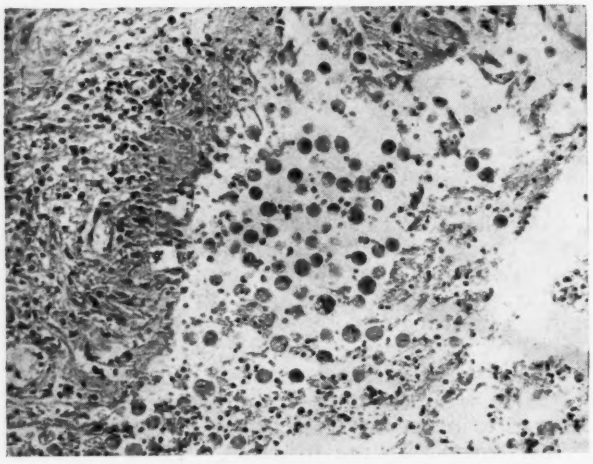


FIG. 428.—Showing emœbæ in margin of ulcer—active forms. (× 150).

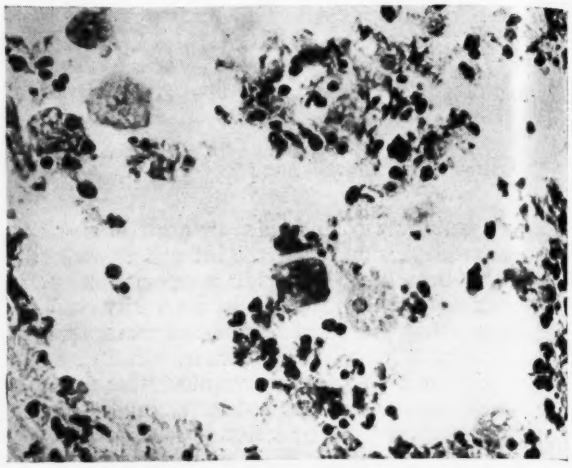


FIG. 429.—Same as *Fig.* 428, showing more clearly the active forms of *Entamœba histolytica.* (× 400.)

FIG. 430.—Photograph of the ulcer before treatment.

FIG. 431.—Showing the deep excavating nature of the ulceration.

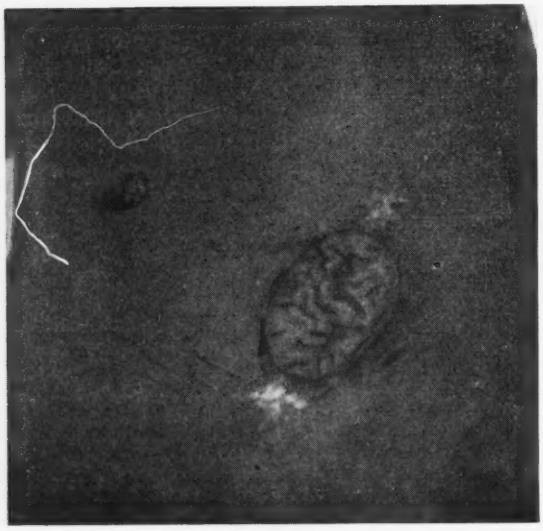


FIG. 432.—The colostomy.

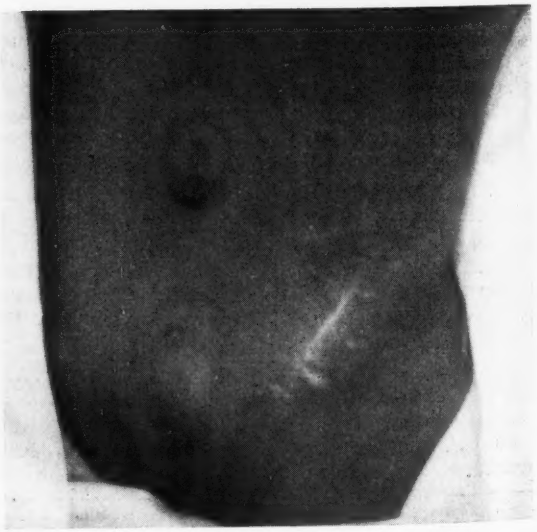


FIG. 433.—After intraperitoneal closure of the colostomy.

The present complaint started one month ago as a small "pimple" on the left buttock. This increased in size and became painful, the pain being of a throbbing nature. He was given an ointment by a doctor and shortly afterwards the swelling "burst", discharging pus. The resulting ulcer apparently has been increasing in size rapidly and the patient was losing weight markedly by the time he was admitted to this hospital.

FIGS. 434, 435.—Showing complete healing.

ON EXAMINATION.—His temperature was 99·4° F., pulse 104, respirations 22. There was evidence of dehydration and loss of weight. Local examination revealed a large ulcer of the left buttock, extending obliquely across the sacrum to the opposite buttock, about 5 in. in length and 2 in. wide. The edge was raised, but not undermined, sloping into the floor of the ulcer, which was covered with dirty, sloughing granulation tissue. The surrounding tissues, for about 1½ in., were swollen and indurated. The nearest point to the anus was about 2 in. The ulcer and surrounding tissues were extremely painful and tender to the touch. The regional lymphatic glands (left inguinal) were moderately enlarged and tender.

Examination revealed a few rales at the base of the right lung. Heart, abdomen, C.N.S. were normal. The blood-pressure was 120/80.

Genito-urinary history and examination were negative.

Laboratory Examinations (March 22).—Complete blood-count: R.B.C.'s 4,870,000, C.I. 1·01, Hb 98 per cent. Leucocytes 15,300; polymorphonuclears 74·5 per cent, large mononuclears 1·0 per cent, lymphocytes 22·5 per cent, eosinophils 1·5 per cent, basophils 0 per cent, metamyelocytes 0·5 per cent. The red cells showed slight anisocytosis, but otherwise no significant changes. The polymorphonuclears showed a slight shift to the left and evidence of early toxic granulation. W.R. was negative.

TREATMENT.—When seen in the ward the condition looked so like a breaking down carbuncle, that treatment for this condition was instituted—urea dressings and short-wave therapy. The condition did not respond, but the ulcer rapidly increased in size. Thin muco-purulent fæces were constantly escaping per anum and contaminating the ulcer area.

On March 26 sigmoidoscopy was performed, revealing a large, necrotic ulcerating area covering the posterior rectal wall. The colon could not be examined. A wedge was resected out of the wall of the ulcer. Section of this specimen showed the presence of an ulcer, in the wall of which amœbæ were found (South African Institute of Medical Research) (*Figs.* 428, 429). Examination of a stool on March 29 revealed the presence of ova of *Ascaris lumbricoides* and flagellate forms of *Trichomonas hominis*, pus, mucus, and red cells, but no amœbæ.

On account of the sloughing condition of the rectum and the contamination of the ulcer, a left inguinal colostomy was done on April 3 in order to divert the contents of the colon. Treatment with emetine hydrochloride was commenced—½ gr. b.d. for twelve days. There was an almost immediate and miraculous response. Concomitantly chiniofon instillations (2½ per cent) in both proximal and distal loops of the colostomy were given, as well as local chiniofon dressings.

The temperature returned to normal, appetite and sleep improved, and there was immediate evidence of healing and retrogression of the ulcer. On May 21, six weeks after commencing emetine therapy, a full-thickness graft was applied to the now cleanly granulating area. A second course of emetine was started on May 23. A portion of the graft unfortunately sloughed, but sufficient took to ensure healing of the ulcer. On July 25 the colostomy was closed, using the intraperitoneal method, followed by another course of emetine for ten days, plus a course of emetine bismuth iodide. The patient was discharged on Sept. 8 healed except for a slight discharge from the colostomy wound, which healed completely subsequently. When seen again in the out-patient department the patient was quite well, had put on weight, and generally had recovered from his malady.

COMMENT

Under certain conditions, *E. histolytica* may invade the skin and subcutaneous tissues of the buttocks and peri-anal regions, causing acute, rapidly spreading ulceration with marked constitutional disturbance. Spread is probably by continuity from infection of the rectum, or via the lymphatics, rarely by the blood-stream. The local lesion is slightly raised, with steep, at times undermined edges, surrounded by an area of induration showing all the characteristic signs of inflammation. The floor of the ulcer is covered with sloughing granulation tissue, the whole entity being extremely painful and tender. Any condition around the anus

conforming to this pattern should warn the medical attendant of a possible amœbic infection of the lower bowel—examination of the stools and a biopsy are necessary for final diagnosis. The response to emetine is miraculous, and this can be used as an effective therapeutic test.

SUMMARY

1. A brief review of the literature on amœbiasis cutis is given.

2. An interesting case of amœbic granuloma of the buttocks is discussed.

3. In acute ulcerative peri-anal conditions not responding to usual chemotherapeutic methods, a therapeutic test of emetine hydrochloride is justifiable. Stool examinations and biopsy for *E. histolytica* should be done.

The authors wish to thank Dr. F. K. Mills, Superintendent of the Johannesburg General Hospital, for permission to publish the case; and Dr. Simpson and Mr. Brandt, the former for making available a microscopical section, and the latter for the microphotographs, both of the South African Institute of Medical Research; also Dr. G. S. Chenik for kindly taking the photographs.

REFERENCES

CRAIG, C. F. (1944), Tice's *Medicine*.
CRAWFORD, quoted by CRAIG, *Ibid.*
ENGMAN, M. F., and MELENEY, H. E. (1931), *Arch. Derm. Syph., N.Y.*, **24**, No. 1, 121.
GABRIEL, W. B. (1945), *Diseases of the Anus and Rectum.*
HEIMBERGER, L. F. (1925), *Arch. Derm. Syph., N.Y.*, **11**, No. 1, 49.
MANSON-BAHR, P. (1943), *The Dysenteries.* London: Cassel & Co. Ltd.
— — (1938), *Trans. R. Soc. trop. Med. Hyg.*, **33**, 223.
MELENEY, F. L., and MELENEY, H. E. (1935), *Arch. Surg., Chicago*, **30**, 980.
MCCONAGHEY, R. (1945), *Indian med. Gaz.*, **80**, Feb., 79.
NGAI, S. K., and FRAZIER, C. N. (1933), *Clin. med. J.*, **41**, No. 11 and 12, 1154–70½.
TIXIER, L., et al. (1927), *Ann. Derm. Syph., Paris*, **8**, 521.

TOOTH PLATE IMPACTED IN GULLET FOR FIFTEEN YEARS

REMOVAL BY TRANSTHORACIC ŒSOPHAGOTOMY

BY G. GREY TURNER

CASE REPORT

HISTORY.—The patient, E. C., aged 52, a married woman, was admitted to Hammersmith Hospital in November, 1945, with the story that she had swallowed a denture fifteen years previously. Her memory of the occasion was very vivid and she could confirm the date as Jan. 3, 1931, for it was the same day as a serious accident to her brother-in-law whose leg had to be amputated in consequence. An experience of seven years as surgical registrar in the Newcastle-upon-Tyne Infirmary taught me how easy it is for a patient to be wrong about matters of history, even to the extent of a year or two, so great care has been taken to verify the date in this case, and I am indebted to the patient and to her medical attendant for assistance on this point and with regard to other items of the history.

When the misadventure occurred the patient was at midday dinner, which consisted of roast chine of pork, onion stuffing, and roast potatoes, which she very much enjoyed. During the meal the patient suddenly noticed that her tooth plate had disappeared and exclaimed: "Oh, I have swallowed my teeth". The denture was an upper one with two front teeth; she knew that it was defective and ought to have been repaired, but at the time she was unable to afford to have this done. As she had no pain or inconvenience the patient finished her meal and then completed the weekly washing, afterwards cleaning up the wash-house before she sat down to rest. The doctor was sent for and made an examination of the mouth, but without using instruments; he also felt the neck and told the patient that the teeth were not in the throat. At that time there was no X-ray or œsophagoscopic investigation. She was assured that the teeth would probably pass and, in the hope of aiding matters, was put on a diet of dried bread with very little to drink. This diet was continued for about a week, when she had a very hard and difficult evacuation and was told that the teeth had probably come away, though no special examination of the stool was made. Thereafter she remained quite well and symptom-free for about three months, when she first noticed a pain up the right side of the neck reaching as far as the ear. Again the doctor was consulted, but assured her that the teeth had dissolved and that the pain was probably nervous. After this reassurance the patient felt quite well for ten years, when she gradually began to experience a little difficulty in swallowing. At first this was only associated with solid foods, but it became slowly and steadily worse, and especially so during the past year. For the last two years the patient had only been able to take soft food and had to be very careful in swallowing. She also had pain on the right side, said to be due to pleurisy and was in bed about three weeks and had cough but never brought up blood. During the summer of 1944 she had various symptoms of discomfort in the chest and upper abdomen attributed to the menopause. There was pain in the epigastrium and sickness. About Jan. 2, 1945, the patient was taken very ill with pain across the lower chest and in the upper abdomen, but this was not obviously connected with swallowing. A surgeon who was called in was not satisfied with the diagnosis of gall-stones which had been made. When doubt was expressed about the diagnosis the patient volunteered the information that she had previously swallowed her tooth plate and asked if her doctors thought this might have anything to do with her trouble. Because of this disclosure a radiograph was taken, which showed a dark shadow in the thoracic œsophagus which might have been a foreign body, but a throat specialist declared that all was in order. In spite of this finding the patient went into hospital for an abdominal operation early in June, 1945. The gall-bladder was found to contain small stones and was removed. The patient made a good recovery and returned home, but after a week or two had worse trouble with swallowing than ever before. Both liquids and solids seemed to stick just behind the middle of the breast bone and produced great pain. The pain on swallowing was also very bad and the patient could only get relief by bringing up whatever she had attempted to swallow. The regurgitated material was unaltered food. In the mornings before taking food

she brought up quantities of mucus. The doctor prescribed medicine which sometimes eased the patient, but not always. The retrosternal pain, sickness, and malaise persisted. In retrospect the patient realized that during the last three years she had lost ground, and about 2 st. in weight, and often had to ask assistance with her household duties. Her new medical attendant, Dr. J. R. Baker, of Winterton, in Lincolnshire, the doctor whom she originally consulted having died, began to wonder if the teeth really were still impacted in the œsophagus and if they might be the cause of her pain. As a result of talking over the matter with the author, it was decided that the patient should come to Hammersmith Hospital for investigation, and she was admitted there under my care.

ON ADMISSION.—She was a slightly built woman about 5 ft. in height, of calm demeanour, and apparently in reasonably good health. While not obviously robust, she was of the wiry type and throughout proved herself most co-operative and helpful. Her only complaint was of difficulty in swallowing, with some pain and soreness on the right side of the neck extending from the clavicle to behind the ear. She was also somewhat concerned that she felt rather weakly and continued to lose weight. Pulse and temperature were normal and a full ordinary examination did not elicit any abnormal physical sign.

FIG. 436.—Radiograph showing what was taken to be a fixation wire on the tooth plate. It was situated opposite the body of the 9th dorsal vertebra.

A plain X-ray examination made the day after admission showed a linear opacity lying obliquely in the position of the œsophagus at the level of the 9th thoracic vertebra (*Fig.* 436). The opacity was about ½ in. long, about the thickness of a piece of dental wire, and slightly curved. In fact, it had just the appearance of a fixation wire on a dental plate, but neither teeth nor plate were seen. Dr. Duncan White tells me that on re-examination he thought he could discern the shadow of two teeth, but there was certainly no delineation of a tooth plate which could be detected by an ordinary observer. The administration of an opaque medium confirmed the relationship of the foreign body to the œsophagus and at the site the bolus of medium was distorted. Thus the appearances were compatible with a tooth plate with a fixation wire impacted in the œsophagus. At this time the patient was able to take soft food apparently without difficulty, but she was very careful during the act of swallowing.

On Dec. 3, Mr. Ivor Griffiths made an œsophagoscopic examination, using intravenous anæsthesia. The upper part of the gullet was normal, but at the position indicated by the X-ray shadow there was a slight constriction (as was said, but not recorded, "the appearances were those of stenosis, but with a wide mouth"). Just beyond was a mass of granulations which bled very freely on the slightest touch. Amidst this granulating area the sharp edge of a vulcanite tooth plate could just be seen, though it was almost at once obscured by bleeding. The actual teeth were not visible. With a suitable forceps the edge of the plate was grasped, but it was firmly fixed at the site of impaction, for it could not be drawn upwards or pushed down, though it could be very slightly moved. It could not have been dislodged, at least upwards, without using great and unwarranted force. It seemed certain that the foreign body was lying in an ulcerated area and that the œsophageal wall was softened and friable and might readily be torn or perforated by some unguarded movement of the hard substance. The question of breaking the plate and removing it in fragments was considered, but the least manipulation brought on free bleeding and with vivid pictures in mind of several examples of injury to the aorta by foreign bodies it was concluded that any such attempt would be most unwise. The patient was just a little upset by this very careful and restrained examination. She felt slightly cold, had slight pyrexia, and experienced more tenderness on swallowing for a day or two. A further radiograph made three days afterwards showed the wire a little lower than before and so altered in contour as to suggest that the tooth plate had been slightly rotated. There was a more marked degree of obstruction to the passage of a swallowed opaque medium. Meanwhile, I had been giving thought to the proper management of the case and had decided to carry out œsophagotomy, using a left-sided transthoracic approach. The consideration which led me to this conclusion are set out in the Commentary. Care was taken to prepare the patient. Her general condition was daily improving as the result of hospital routine and feeding, so that from the nutritional point of view it did not seem necessary to carry out preliminary gastrostomy. A course of sulphanilamide and 100,000 units of penicillin were administered.

AT OPERATION.—The operation was carried out on Dec. 11. The intratracheal method of anæsthesia with gas, oxygen, and ether was carried out by Dr. Woodfield-Davies, and as usual was most satisfactory. The incision was made in the seventh interspace, extending from the edge of the sternum right back to the edge of the erector spinæ. The exposure after rib-spreading was satisfactory and it was not necessary to divide ribs. The general pleural cavity was normal looking and entirely free from adhesions. The descending aorta seemed to be longer and much more prominent than usual and completely obscured the œsophagus, and the sulcus on its inner side between this vessel and the œsophagus was so deep as to be very striking. As a first step, the inferior pulmonary ligament was divided. The œsophagus was then easily exposed and palpated and the foreign body located just a little below the lung root. The pleura overlying this part of the œsophagus was slightly reddened and a little thickened; it was incised vertically from just above the diaphragm to a point about an inch below the site of impaction of the plate. It was somewhat thickened at the upper end of this incision and the cellular tissue between it and the œsophagus was firmer in texture than normal, and seemed to bind the tissues together. At no point was the œsophagus adherent to the aorta and separation of the œsophagus from its surroundings was easy. At the site of impaction the œsophagus felt hard and simulated a malignant new growth, though just below this area it was actually a little dilated with a rather softer wall than normal. The œsophagus was bluntly mobilized by the finger and

elevated from its bed with the help of a ring forceps. The cellular tissue was protected by gauze wrung out of flavine solution and the œsophagus opened between guide sutures introduced into its wall just below the level of the impacted foreign body. The incision was about 1½ in. long and planned so that its upper extremity was situated ½ in. below where the foreign body lay. I was afraid that if carried higher it would extend into an ulcerated area surrounding the tooth plate and that this part might not heal satisfactorily. Except for some thickening from œdema, the œsophageal wall was apparently normal, and on opening its lumen there was only a little mucus, easily wiped away.

The finger, passed up the œsophagus, disclosed a slight narrowing just below the ulcerated area in which the plate was lying, but the edge of the latter could readily be felt as the tip of the finger easily went through the narrowed area. With a pair of angulated necrosis forceps the plate was grasped, and though it could not

FIG. 437.—The tooth plate after removal. (× ⅘.)

immediately be drawn down, it was slightly rotated and then easily manipulated free and removed. There was no bleeding and no free escape of purulent discharge and, in fact, nothing but what could be readily mopped away without soiling the parts.

It had originally been intended to suture the incision in the œsophagus in the transverse direction after the principle of pyloroplasty and a suture was placed through the upper and lower angles of the incision as a first step in this stage, but the ends of the incision did not approximate readily and, in fact, the upper one could not be drawn down at all. The suture was therefore removed and the incision carefully closed vertically. The mucous membrane, which seemed unusually voluminous, retracted quite considerably, but four interrupted sutures, passed from within the lumen, brought its edges together. The muscular wall, which was twice as thick as usual, was then carefully approximated by half a dozen lightly tied sutures, i.e., lightly tied in order not to cut through the softened and œdematous muscle. About six other sutures were used to bring together loose tissue over the site of the incision but the cellular tissue was scanty, probably due to the peri-œsophagitis. When completed the closure appeared to be entirely satisfactory. The end of the last stitch was left uncut and a drainage tube ⅜ in. in diameter, and without side holes, was threaded along it until it reached the lower end of the œsophageal incision. The idea was to provide a direct track to the surface in the event of leakage. A powder of sulphanilamide and flavine was scattered around about the area.

It had been intended to draw the posterior pleura over the sutured œsophagus, but that structure proved to be friable and, what is more, was rather fixed and would not come readily together, so perforce had to be left open. The chest was carefully closed. The tube was brought out at the extreme anterior end of the parietal incision, which it was judged would be the most dependent spot with the patient in the sitting posture. At the end of the operation it was observed that there was no sucking of air either through the tube or by its side. Penicillin, 10,000 units, was instilled along the tube. There were no unusual features during the operation and the patient tolerated the intervention without causing the slightest anxiety.

PROGRESS.—The after-progress was entirely uneventful, it was almost afebrile, and the pulse, which was never quicker than 105, was normal by the end of the week.

On the day following operation 100,000 units of penicillin was given by the intramuscular route and sips of a penicillin solution were given. Nothing else was allowed by the mouth for four days; during this time she had fluids both intravenously and per rectum. Thereafter mouth feeding was gradually advanced and by the end of a week she took a sufficiency of semi-solid food and by the end of the second week ordinary careful diet, and thereafter

FIG. 438.—Radiograph of plate after removal. It shows the buried wire.

the full diet of the hospital. There was never any discharge from the tube and no moisture; it was removed on the eighth day. The incision in the parietes healed *per primam*.

On the first day of January, 1946, i.e., just three weeks after operation, X-ray examination showed some slight dilatation of the distal portion of œsophagus, but there was no sign of obstruction. By that time the patient was swallowing normally, feeling in every way quite well, and was gaining weight. She left the hospital exactly 22 days after operation and travelled to her home, a distance of 129 miles, by car. Since then she has steadily improved and considers that her health is quite restored to normal.

In April she was reported as very well and had gained 17½ lb. since operation. She was doing her own housework and eating with relish things she had not touched for years. In November the patient reported that she was perfectly well in every way.

The plate was of the shape and size shown in *Fig.* 437. Its length was 1⅜ in., width 1⅛ in., and greatest depth ⅞ in. (The average transverse diameter of the œsophagus at the site of impaction is usually regarded as 1⅛ in.) Except that it was completely blackened, it was apparently unchanged as the result of its long sojourn in the œsophagus. The wire, seen in the films taken *in vivo*, is embedded in the material of which the plate is made. It weighed 36 gr. and appeared to be of the ordinary vulcanite composition, but no analysis has been made. As the plate may be said not to have shown in the body it was X-rayed, with the result shown in *Fig.* 438.

COMMENTARY

In this case I felt it wise to approach the problem from the point of view of pathology and assumed that most likely the tooth plate had caused

extensive pressure necrosis in the œsophagus and was lying in an ulcerated area which might even be involving the adjoining aorta. In such circumstances it seemed unlikely that the plate could be safely removed endoscopically because of the way in which it was probably embedded, and in consequence there would be considerable risk in that form of removal which might result in serious trauma precipitating disaster. My first view was, therefore, that the foreign body should be approached by a direct incision into the œsophagus under the unimpeded guidance of the eye, using the transthoracic route.

Because I assumed that there would be a very considerable degree of sepsis about the tooth plate, with risk of infection of the cellular tissue and the equally striking, rendering the route most uninviting (*Fig.* 439). At the same time it must be recognized that the subject was one of those very rigid formalin-prepared bodies where all the structures are much hardened and to add to the trouble the left lung was almost solid. In life I am sure the difficulties would be somewhat less than we encountered, though there is no way that I can imagine of overcoming the obscurity produced by the aorta and the great depth of the œsophagus from the surface. I therefore reverted to the idea of a transthoracic approach, which had the great advantage of familiarity as well as accessibility.

Before finally deciding to deal with the problem by this route it was considered that despite the long period of impaction and the probable ulceration we

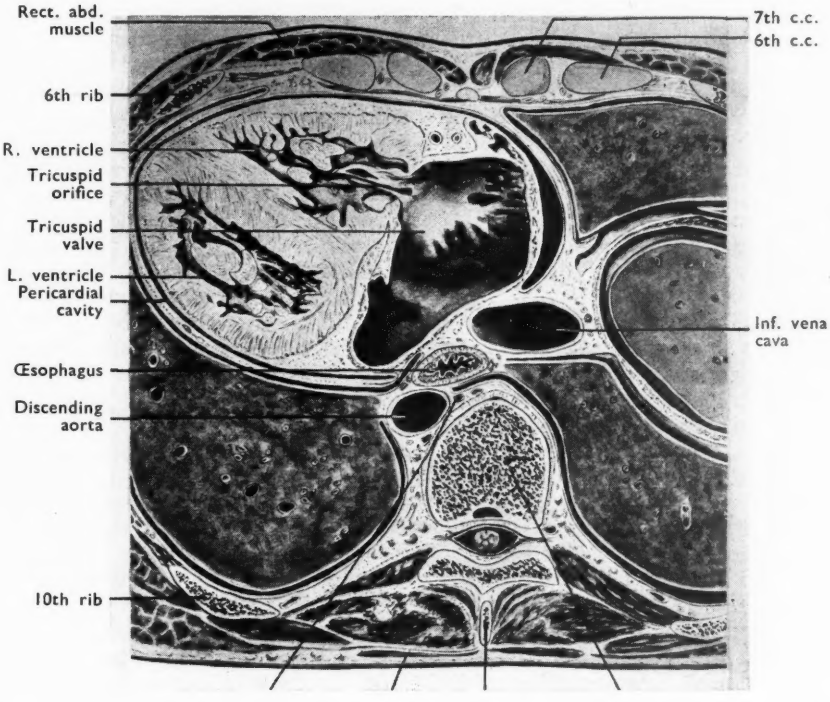


FIG. 439.—Transverse section of the thorax through the 9th dorsal vertebra, showing the depth of the œsophagus from the posterior surface. (*From Symington's "Atlas of Topographical Anatomy".*)

pleura, I next had in mind the approach to the œsophagus by the posterior mediastinal route. There was also the fear that during removal the œsophageal wall might be torn and would not heal satisfactorily, and that a fistula would be a probable sequel. For these reasons I assumed that drainage had better be directly posterior rather than across the pleural cavity. Working on the cadaver, I again tried out the posterior mediastinal approach from the *left* side. This emphasized the difficulties of this route due to the depth of the œsophagus, which was about 4 in. from posterior skin surface, and also the way that the œsophagus was obscured by the great prominence of the aorta.

On another occasion I got my assistant, Mr. D. M. Douglas, to try out a similar approach from the *right* side on the same cadaver. Although the prominence of the aorta was no longer a barrier, the depth of the œsophagus from the surface was ought to look at the foreign body endoscopically to determine if the plate could perhaps be extracted that way—if not unexpectedly easily, at least without undue difficulty and trauma. This plan was especially considered because on the X-ray films only the gold band could be seen, the tooth plate itself not casting any shadow, and I thought it possible that part of the plate had broken away, thus rendering its removal easier. As the record shows, the plate was firmly impacted and it was not considered safe to try to remove it by withdrawal upwards. It might have been assumed that the plate could have been drawn downwards if the stomach had been opened and forceps introduced into the œsophagus from below, but this is a blind method by which any untoward complication like perforation or hæmorrhage could not be properly dealt with. Of course, it must be remembered that the œsophagoscope can be used through a gastrotomy.

Though these considerations supported the decision to carry out thoracotomy, the question of the side to be employed arose. For operation for neoplasms of the œsophagus situated at about the site of impaction of the plate, the approach from the right side is much the better, as was urged by my old pupil, the late Laurence O'Shaughnessy (1934) and further emphasized by the experience of my senior assistant, R. H. Franklin (1942). But, confronted with the problem of a foreign body which had been so long impacted, there was the haunting fear of involvement of the aorta, and it was felt that any complication thus arising could be so much better dealt with under the guidance of the eye from the left-sided approach.

The question of preliminary gastrostomy had to be reviewed, but I did not think of adopting this measure in the case under consideration because the patient was at least able to swallow liquids and semi-solid food without much difficulty and was consequently in reasonably good condition. But it would have been quite a sound plan to make a gastrostomy had it been necessary to improve the nutrition, and, at the same time, it would have sufficed to solve the feeding difficulties and anxieties after operation and it would also lessen the infective factor by diversion of septic particles from the site. It would have the further value that the incised and sutured area of the œsophagus would be protected from strain during healing. Though it would not have been wrong to make a preliminary gastrostomy even without such indications, I had sufficient confidence in the healing properties of the œsophageal wall to feel that it could be dispensed with. Of course, gastrostomy could very readily have been carried out as a last step in the operation of removal if called for by unexpected circumstances, such as the unusual friability of the œsophageal wall or uncertainty as to the efficiency of the suture line.

When considering the possible difficulties of the actual operation against which I felt one ought to be forearmed, the most serious concerned the relationship of the foreign body to the aorta. From clinical experience and a knowledge of pathological material and of the literature (Grey Turner, 1946), I knew only too well that there is a remarkable tendency for sharp objects to erode towards or into the aorta, and it occurred to me that during the process of removal that vessel might be damaged, possibly the last stages of an impending perforation being made actual. Should this occur I thought the safest plan would be rapidly to separate the aorta from the œsophagus and then endeavour to close any hole in that artery by direct suture. I have some little experience of this matter in cases where perforation of the aorta has occurred during an attempt at œsophagectomy. In such circumstances I have been able to arrest hæmorrhage successfully by drawing the edges of a small aperture together, using a sharp-pointed cleft-palate needle with fine catgut, such sutures of apposition being reinforced by an additional stitch or two in the adventitia. Any persisting oozing from the site might be controlled by the application of fibrin foam, although this material was not available on the occasions in which I had to suture the aorta. Recently surgeons have been fortified by the experience of Professor Crafoord (1945), of Stockholm, who has taught us that the descending thoracic aorta can be compressed for as long as 20 to 25 minutes so that repair of that vessel may be more effectively carried out. With a ragged tear or friable tissue it might even be wise to excise a small section of part of the wall of the aorta in order to get clean fresh edges for suturing.

Another possible difficulty that occurred to me was laceration of the œsophageal wall occurring in the neighbourhood of the foreign body or friability at the suture line. I have actually seen the œsophagus torn completely across at the site of an ulcerating lesion. Probably even at the worst some sort of repair could be made, but it might not be very efficient. In these circumstances I pictured that it would be necessary to intubate the œsophagus and also to bring a tube from the immediate proximity of the sutured œsophagus out through the chest wall to make a track to the exterior in the event of leakage. The point at which the chest wall should be traversed admits of some little discussion. To bring the tube out immediately posterior suggests itself as the nearest and probably the best route, but in carrying this out there one is embarrassed by the aorta (Grey Turner, 1916). The next best thing would be to bring the tube out through the parietes at the anterior end of the intercostal incision, for in the sitting position which the patient would probably occupy after operation, this might prove to be the most dependent spot. If effective suture of the œsophagus turned out to be quite impracticable, then it would be essential to make a gastrostomy after the chest had been safely closed. As a direct consequence of inefficient suturing or of extra friability, a fistula might develop, but the presence of a drainage tube down to the site of repair would probably determine the safest and most convenient external site for the external opening to be situated. Among the complications to be borne in mind when the infected and friable œsophagus requires to be opened, mediastinitis comes first. If symptoms of this condition become apparent as judged by the very ill condition of the patient, together with tender swelling and possibly crepitant œdema at the root of the neck or air bubbles demonstrated in the mediastinum by X rays, then the position would already be very serious, but drainage and the use of penicillin might be sufficient to stave off impending disaster (Grey Turner, 1946). As a late sequel a stricture might develop, but that would probably be the least of the troubles because with such a very definite inflammatory origin its management would only be a question of the intelligent use of bougies.

Finally, it would be unwise to assume that the successful outcome of this case was entirely due to the use of penicillin and the other antiseptic adjuncts now available.

REFERENCE

CRAFOORD, C., and NYLIN, G. (1945), *J. thorac. Surg.*, **14**, 347.
FRANKLIN, R. H. (1942), *Brit. J. Surg.*, **30**, 141.
O'SHAUGHNESSY, L. (1934), *Ibid.*, **22**, 365.
TURNER, G. GREY (1916), *Brit. J. Surg.*, **3**, 552.
— — (1946), *Injuries and Diseases of the Œsophagus*. London: Cassell.

CAROTID BODY TUMOURS

By H. M. GOLDBERG

DEPARTMENT OF SURGERY, MANCHESTER ROYAL INFIRMARY.

Because of their rarity carotid body tumours are very seldom diagnosed before operation. When met with unexpectedly at operation they present difficult technical problems. Most of the full accounts of the condition have appeared in the American literature. Bevan and McCarthy collected 143 cases in 1929. Gratiot reviewed the subject in 1943 and found 253 cases reported in the literature. In this country Hutchinson first drew attention to "peculiar tumours" in the neck which he called "potato tumours", but he did not associate them with the carotid body. Gilford (1904), Stewart (1931), and Gordon-Taylor (1940) reported three, four, and five cases respectively.

CASE REPORTS

Case 1.—A woman, aged 23, had noticed a swelling on the right side of her neck for two years (*Fig.* 440). This had gradually increased in size. She complained of frequent sore throats and occasional spontaneous fainting attacks. These had occurred on the average once a fortnight. On one occasion she had lost consciousness when the lump was accidentally touched.

On Examination.—A firm smooth swelling $2\frac{1}{2} \times 2\frac{1}{4}$ in. was found half in front of the right sternomastoid, half deep to it, at and above the level of the upper border of the thyroid cartilage. It was mobile from side to side, but not vertically, and showed definite transmitted pulsation. No murmur was present on auscultation. Pressure on the common carotid below the tumour caused the pulsation to cease and the tumour to shrink from $2\frac{1}{2} \times 2\frac{1}{4}$ in. to $1\frac{3}{4} \times 2\frac{1}{8}$ in.

On pressure over the tumour the patient became pale and clammy within twenty seconds. Her respiration quickened and her eyes began to roll. She began to cry —without any cause, as she stated later—and her face twitched. Within a minute she had lost consciousness. The blood-pressure, which at rest was 130/75 mm. Hg, fell to 80/60 mm. Hg and quickly recovered on release of pressure. Her pulse slowed only insignificantly. When the pressure was more prolonged the radial pulse disappeared altogether. Pressure on the common carotid or the left carotid sinus caused no change.

Electrocardiography (*Fig.* 441).—Sinus arrhythmia at a rate of 70/80. Pressure on the tumour has very little effect on the rate, but causes the P waves to disappear. This may indicate the onset of nodal rhythm.

Electro-encephalography (*Fig.* 442) (Mrs. J. Turner).—A routine E.E.G. examination was first carried out with the patient at rest in a reclining position. A Grass, 3-channel ink-writing electro-encephalograph was used and bipolar recordings were taken from twelve symmetrically placed electrodes. Occasional low-voltage

Fig. 440.—*Case* 1. Photograph showing the swelling in the right side of the neck.

waves at 4–5 c/s appeared, but otherwise the record was within normal limits, showing a moderately sustained alpha rhythm at 10–12 c/s of about 40–50 microvolts.

A right-sided anteroposterior electrode arrangement was then used, and the effects of compression were observed. Pressure on first the right and then the left carotid artery proximal to the tumour, and also on the left carotid sinus, showed no change in the E.E.G. pattern.

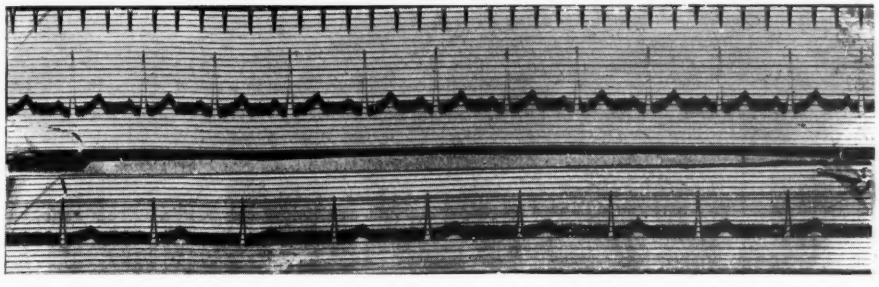


Fig. 441.—*Case* 1. E.C.G. Upper tracing before compression of the tumour. Lower tracing after 10 seconds' compression.

Compression of the tumour showed a sudden appearance of widespread irregular high-voltage slow waves at 1–3 c/s after 17 seconds. Pressure was maintained for 45 seconds in all, and the slow waves persisted for a further 15 seconds after release, then ceased abruptly, the E.E.G. pattern reverting to the resting picture.

Immediately prior to compression of the tumour the resting pulse-rate was 108, but three seconds before the onset of the slow waves it had fallen to 48. Towards the end of the discharge no pulse was palpable, but 10 seconds after the reappearance of the normal pattern it could just be felt. Twenty-five seconds after this the rate had risen to 120 per minute.

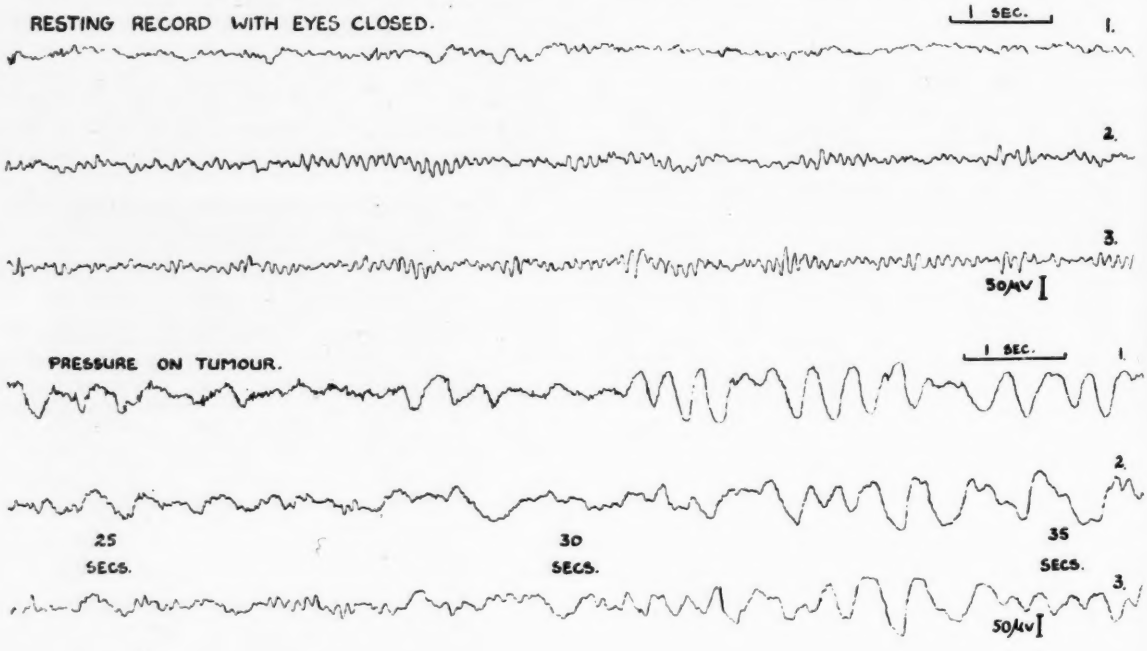
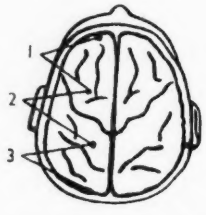


Fig. 442.—*Case* 1. Electro-encephalographic records (*see* text).

The experiment was repeated using a left-sided anteroposterior electrode arrangement and a similar result was obtained.

BIOPSY.—On Dec. 29, 1945, a biopsy was performed by Mr. R. L. Newell through a transverse incision. The vascularity of the tumour was striking. No change in blood-pressure or pulse-rate were noted on compression of the tumour under the anæsthetic (gas, oxygen, ether).



The right common carotid artery was compressed daily, at first for 5 minutes, increasing gradually to 35 minutes. No abnormal physical signs were detected in the nervous system after 35 minutes' compression.

OPERATION (Jan. 31, 1946) (Mr. R. L. Newell).—anæsthetic: gas, oxygen, ether. The incision extended from behind the angle of the jaw to the sternoclavicular joint, with a slight convexity forwards. The internal jugular vein had to be divided. The tumour was not unduly adherent to surrounding structures apart from where the previous biospy had been performed. In trying to dissect the tumour off the common carotid, which was completely encircled by it, the vessel was opened and had to be tied.

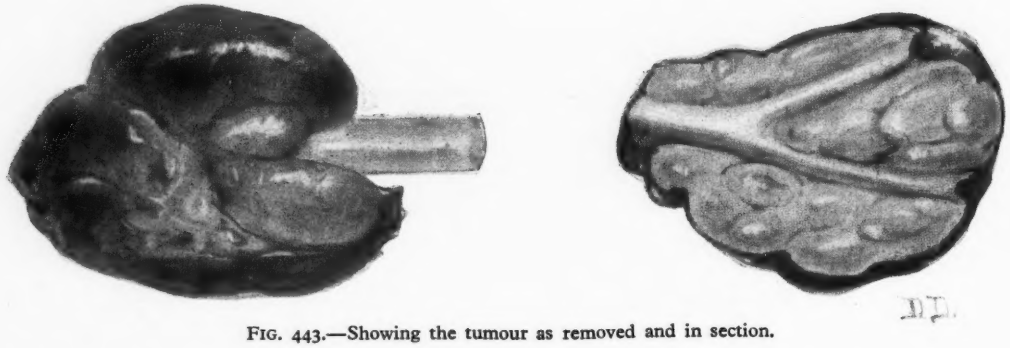


FIG. 443.—Showing the tumour as removed and in section.

The post-operative course was uneventful. No important nerves were damaged and no signs of cerebral anæmia were observed. Three months after operation the patient showed no ill effect from the carotid ligation and was able to carry out her normal occupation.

PATHOLOGICAL REPORT (by Dr. J. Davson, Victoria University, Manchester).—Macroscopically, the specimen consisted of a somewhat pear-shaped mass with an indistinctly lobulated outer surface, measuring 5 cm. × 3·5 cm. (*Fig.* 443). It weighed 30 g. On section the mass is of a fairly uniform whitish colour, with slaty blue and yellowish foci visible. The common carotid enters the tumour at the lower pole and divides into the internal and external carotid branches, which leave the tumour from its superior aspect.

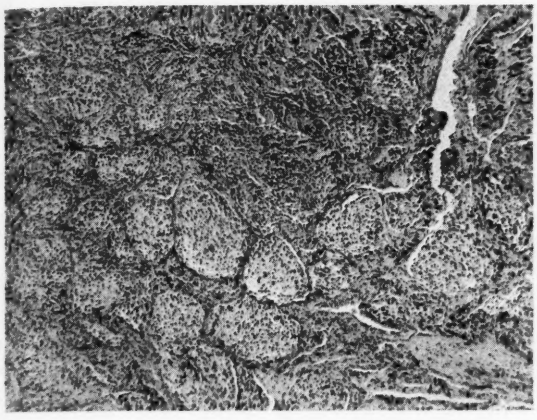


FIG. 444.—*Case* 1. A low-power view showing the typical glomerulus-like cell groups in the lower central field, while top centre the cells show an ill-defined trabecular arrangement. Hæmatoxylin and eosin. (× 30.)

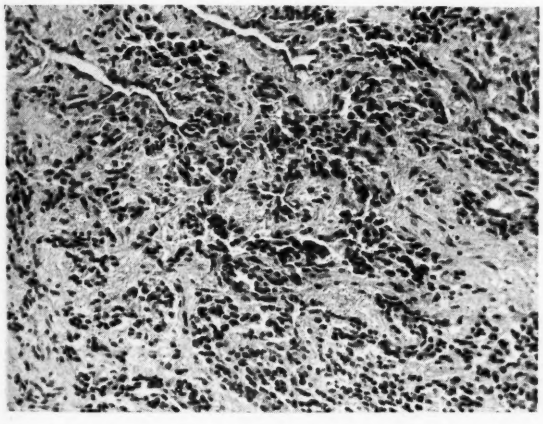


FIG. 446.—*Case* 1. A higher magnification of the trabecular type of cell arrangement, showing a predominance of hyperchromatic nuclei. The cell cytoplasm tends to be darker staining. Hæmatoxylin and eosin. (× 120.)

ON SECTION (*Figs.* 444–446) there is some variation in the histological appearance of the tumour, both in the arrangement of the cells and in their appearance. The characteristic cells have pale-staining round or oval nuclei with well-developed nuclear membrane and a coarse chromatin network. The cell outline is indistinct and polyhedral in shape, the cytoplasm is pale-staining and mainly granular. The cells are arranged characteristically in round compact masses about the size and shape of renal glomeruli and are separated by compressed vascular spaces lined by prominent capillary endothelial cells. In other areas the cells form much smaller groups and cords which are separated by a meshwork of capillaries lined by hypoplastic endothelium.

Elsewhere the cells have darker-staining oval or round nuclei and a more eosinophilic cytoplasm, and the arrangement is in somewhat irregular-shaped ill-defined cell masses.

The stroma in some cases consists of acellular fibrous tissue, in other areas it is represented only by the flattened capillary spaces. A thin fibrous capsule is present.

Stains for chromaffin substance were negative.

DIAGNOSIS.—Perithelioma of the carotid body.

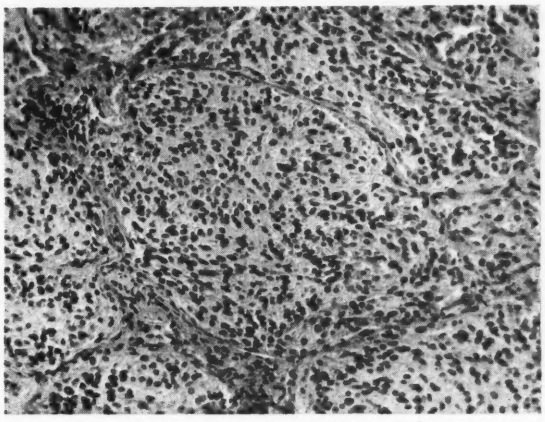


FIG. 445.—*Case* 1. A higher magnification of a glomerulus-like body, separated from adjacent similar bodies by flattened vascular spaces. Some nuclei are vesicular in type, others are hyperchromatic. The cell cytoplasm is pale. Hæmatoxylin and eosin. (× 120.)

Case 2.—Mr. M., aged 37, a lecturer in architecture, came under the care of Mr. W. R. Douglas in June, 1940, complaining of a swelling in the left side of the neck of two years' duration. It was slightly sensitive to pressure and had varied in size. The patient gave a history of rheumatic fever at 18 and a winter cough for several years.

ON EXAMINATION.—There was a mass deep and anterior to the left sternomastoid muscle at the level of the angle of the jaw. It was smooth, tender, not unduly fixed, and of firm consistency. The spleen was palpable ½ in. below the costal margin. The blood-pressure was 140/90 mm. Hg, and the Wassermann reaction was negative. A pre-operative diagnosis of tuberculous lymph-glands was made.

AT OPERATION (July 4) (Mr. W. R. Douglas).—Under avertin-gas-oxygen anæsthesia a vertical incision was made along the sternomastoid. The muscle was split and "a very vascular looking tumour lying behind the bifurcation of the carotid" was exposed. It was here that the true nature of the tumour was recognized. The vessels were closely adherent to the tumour and had to be ligated.

Next morning there was no paralysis. Thirty-six hours after operation, however, the right side of the face, the tongue, and the right arm were paralysed. Aphasia and dysphagia were marked. The right leg was unaffected. On the next day (July 7) some improvement was noticed and this continued until the patient was discharged on Aug. 6, when only slight facial weakness remained. The blood-pressure on discharge was 125/80 mm. Hg. A year later no residual weakness remained and the patient had returned to his previous occupation. He has been seen at regular intervals ever since and has been keeping in perfect health.

HISTOLOGY.—The tumour cells are arranged partly in well-defined spherical masses separated by broad bands of acellular collagen, and partly in less well-defined groups separated by irregular spaces lined by endothelial cells.

The cells are of polygonal outline, with fairly eosinophilic cytoplasm. The nuclei are mainly hyperchromatic, predominantly oval, and occasional multinucleate forms are present. No definite mitoses were seen, but the cytological appearances are suspicious of a low-grade malignancy (*Figs.* 447, 448).

DIAGNOSIS.—Perithelioma of the carotid body.

DISCUSSION

Historical.—The carotid body was first described by v. Haller in 1743. In 1880 Riegner removed the first carotid body tumour. He ligated the common carotid artery. The patient developed hemiplegia and died on the third day. Maydl's case in 1886 was the first to survive. A temporary hemiplegia followed ligation of the arteries. The first removal without ligation was carried out by Albert in 1889.

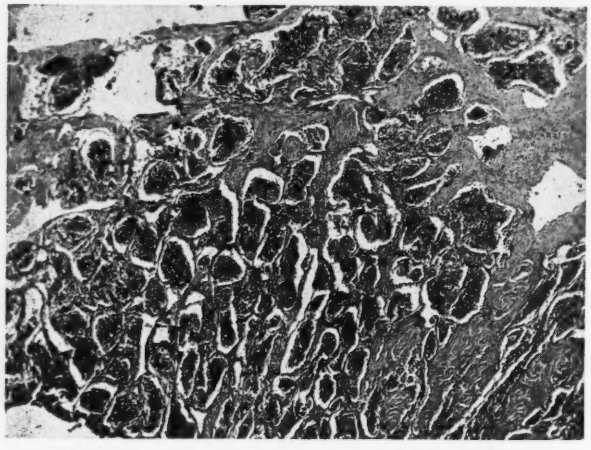


FIG. 447.—*Case* 2. A low-power view showing the glomerulus-like bodies separated by strands of hyaline fibrous tissue. The clear spaces surrounding the cell masses are the result of shrinkage. Hæmatoxylin and eosin. (× 30.)

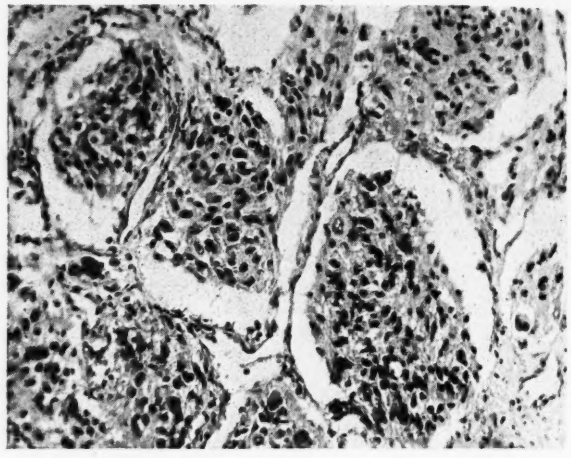


FIG. 448.—*Case* 2. A high-power view showing the glomerulus-like bodies in which the cell nuclei are all hyperchromatic and of somewhat irregular shape. Hæmatoxylin and eosin. (× 120.)

Embryology.—The origin of the carotid body is still under dispute. According to Kohn (1900) it develops from the sympatho-chromaffin cells, which later mature into chromaffin cells. The carotid body has therefore been classed into the chromaffin system, together with the suprarenal medulla and the aortic and coccygeal bodies. Kohn's view of the affinity of the cells of the carotid body to chromic salts has not been confirmed in the human, although it holds good with other species in which the carotid body contains a larger element of sympathetic ganglion cells. According to Maurer (1895) and Boyd (1937) the carotid body develops from the mesoderm of the third branchial arch and receives early connexions from the artery of the arch and the ectoderm of the glossopharyngeal nerve, which is the nerve to the third branchial arch. Elements from the sympathetic and vagus are incorporated at a later date.

Anatomy.—The carotid body is inconstant in man. Funke (1904) found a carotid body in only one out of eight fœtuses dissected, and that only on one side. The bodies increase in size up to the age of puberty and then slowly atrophy.

The carotid body is situated behind the common carotid artery at its bifurcation. It is reddish brown in colour, oval in shape, and measures up to 7 mm. in its long axis. It is surrounded by a firm capsule which sends septa into its substance, subdividing it into lobules. The connective tissue is rich in medullated and non-medullated nerve-fibres. The lobuli consist of spherical masses of large polyhedral cells which contain large eccentrically placed and deep-staining nuclei. The cell membranes are often difficult to distinguish.

The vascular supply is usually derived from the external carotid artery via a small fibro-adipose pedicle called the ligament of Mayer. The artery enters the lower pole and breaks up into branches, each of which enters a lobe and forms a dense capillary network around the cells. The veins form a plexus over the surface of the body, from which the blood is led away by several branches which drain into the superior laryngeal, lingual, and pharyngeal veins. The nerve-supply comes from the glossopharyngeal nerve, the pharyngeal branch of the vagus, and the superior cervical ganglion.

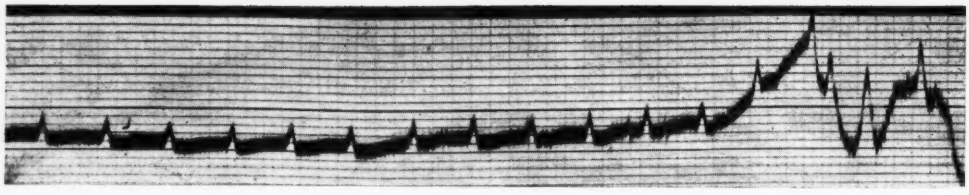


FIG. 449.—E.C.G. in a case of carotid sinus syncope of the cardio-inhibitory type.

Physiology.—Extirpation or absence of the carotid bodies has no adverse effect. Various functions have been assigned to them, but most theories have been disproved. De Castro (1928) was the first to suggest that they may act as chemoreceptors, and Dripps and Comroe (1944) have shown that they respond to chemical changes in the

arterial blood, chiefly decreased oxygen tension and increase in acidity. These stimuli produce a rise of the respiratory and cardiac rates and in arterial blood-pressure, in addition to an increased activity of the cerebral cortex. This mechanism might suggest that the carotid body is derived from the gill cleft mechanism of fish, where it would act as a mediator between the oxygen tension of the water and the respiratory rate. This function of the carotid bodies might therefore be taken to support the theory of the embryological origin from the third branchial arch.

Pathology.—Tumours constitute the only pathological lesion of the carotid body. About 265 cases have so far been reported. They are lobulated and, unless frankly malignant, well encapsulated. They are situated inside the carotid sheath at the level of the bifurcation. As they grow they encroach on the carotid region and may wedge themselves tightly between the mandible and the mastoid process. Inferiorly they may occasionally extend down to the clavicle.

They are slow-growing tumours, usually benign at first, but they may become malignant later. The average length of the history in benign tumours is 4·5 years, that of malignant ones 8·9 years. Benign tumours have been removed after having been present for over twenty years. Some cases must have been malignant from the beginning. Peterson and Mecker (1936) report a case of a woman of 38 with a history of only ten months' duration from whom a malignant tumour was removed. The patient died nine months later of malignant cachexia. A large number of patients in their twenties reported with malignant tumours.

Malignant tumours invade the capsule and involve surrounding structures. Most rapid growth commonly occurs upwards, involving the base of the skull and later the brain. Lymph-gland metastases are rare and only one undoubted case with liver metastases has been reported (Lund, 1917). Two patients of the Mayo Clinic showed signs of possible brain secondaries.

In 50 per cent of cases the tumour completely surrounds the carotid vessels and is inseparable from them. Carotid body tumours are very vascular and have a plexus of veins on their surface, so that if operated upon under a wrong diagnosis the extreme vascularity, as well as their situation inside the carotid sheath at the level of the bifurcation, should correct this error.

Histology.—The majority of tumours arise from the epithelial type of cell. Marchand (1891) and Paltauf (1892) believed that they originate from perithelial cells and suggested the term 'perithelioma'. Although this view has not been generally accepted this term has been widely employed, " as it recalls a prominent structural feature " (Ewing, 1940). A few tumours of nerve-cell origin have been reported (Gordon-Taylor, 1940; Goodsitt and Sudimack, 1945), which are usually described as neuroblastomata, neurofibromata, or ganglion-neuromata.

The cells of the perithelioma are similar to those found in the normal carotid body. The cells often grow in syncytial masses. Where the membrane shows clearly, the cells are polyhedral in shape. The nuclei are hyperchromatic and the protoplasm is granular. Their affinity to chromic salts is inconstant. The dye takes only very faintly if at all. According to de Castro (1928), the carotid body does not contain chromaffin cells.

Harrington, Claggett, and Dokerty (1941), from the Mayo Clinic, describe two patterns, which they call the alveolar and the peritheliomatous. In the alveolar pattern large islands of polyhedral cells are separated from each other by narrow bands of connective tissue. Large thin-walled vascular sinuses traverse the fibrous tissue and are often encroached upon by budding masses of tumour cells.

In the peritheliomatous pattern the alveoli are smaller and more irregular. They are separated from each other by very vascular connective tissue. The cells are arranged in nests or irregular strands. Both our cases belong to this type.

Hyaline degeneration or cavity formation is often seen in the centre of the tumours. In malignant tumours the alveolar formation is lost and the cells grow diffusely throughout. The stroma may become sarcomatous.

Aetiology.—Tumours of the carotid body occur with equal frequency in both sexes. Both sides are equally affected. Four bilateral cases have been reported (Lund, 1917; de Tarnovsky, 1932; Chase, 1933; Harrington, 1941). The average age is 43·2 years (*Fig.* 450).

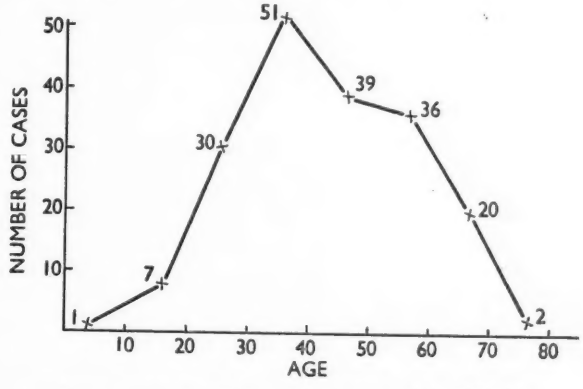


FIG. 450.—Age incidence in 186 cases of carotid body tumours.

Frequent sore throats are often mentioned in the history. The thyroid gland was enlarged in 3 cases (Nix).

Symptomatology.—The most frequent history is that of a painless unilateral slow-growing tumour in the neck. The average duration before the patient consulted the doctor was seven years. Rarely the tumour is sensitive to touch, but it is never painful. Other symptoms are due to compression or invasion of surrounding structures. These are not frequently met with. Hoarseness, dyspnœa, dysphagia, cough, and tinnitus may be due to compression of the vagus. Horner's syndrome, due to pressure on the cervical sympathetic, and earache due to involvement of the cervical plexus, have been reported. Fainting attacks are due to the coexistence of a sensitive carotid sinus. Reid (1920) reported a lemon-yellow pigmentation of the skin in 2 cases which disappeared after operation.

Carotid Sinus Syndrome.—Overactivity of the carotid sinus reflex has been associated with a carotid body tumour in 5 cases out of 260 found in the literature, and in *Case* 1. The incidence of their coexistence is too high to be purely accidental. The nerve-supply of the sinus is similar to that of the carotid body, being derived chiefly from the glossopharyngeal nerve by way of the carotid sinus nerve of Hering and probably also from the vagus, hypoglossal nerve, and the sympathetic. A tumour of the carotid body may therefore occasionally produce an overactive sinus by two mechanisms; either by direct pressure or by way of a reflex arc. Other lesions in the neighbourhood of the bifurcation of the common carotid, such as cervical, lymphadenopathies—chiefly tuberculous—are very occasionally associated with a hypersensitive sinus.

Carotid sinus syncope can be precipitated in subjects with a sensitive sinus by a sudden turning of the head, wearing tight collars, or by pressure over the sinus. It is characterized by pallor, hyperpnœa, muscular twitching, and dizziness, followed by unconsciousness. Flushing of the face occurs on recovery. Two types of carotid sinus syncope are recognized, depending on which of the two efferent pathways—the vagus or the vasomotor depressor nerves—predominates.

In the cardio-inhibitory type in which vagal cardiac inhibition is the predominant factor, a marked fall in blood-pressure is associated with complete asystole or ventricular standstill. The E.C.G. during the attack shows no alteration of potential or only a succession of P waves (*see Fig.* 449). This reaction can often be prevented by atropine.

In the vasodepressor type vasodilatation causes a fall in blood-pressure and the consequent cerebral anæmia results in unconsciousness. The cardiac rate does not slow markedly and vagal stimulation of the heart produces at the most nodal escape, so that the E.C.G. shows either no alteration from normal or absent P waves, as in *Case* 1. This type may often be relieved by sympathomimetic drugs such as ephedrine or benzedrine.

The E.E.G. shows moderate- to high-voltage slow (delta) waves during unconsciousness. This is an unspecific response and can be seen in syncope from various causes such as venepuncture, distension of colon and rectum, etc. (Engel, 1944).

A third type has been described in which syncope is unassociated with any significant alteration in blood-pressure or cardiac rate. This has been called the cerebral type, but its existence is doubtful.

Cure from carotid sinus syncope follows excision of local glands or tumours or follows denervation of the sinus if the hypersensitivity is due to arteriosclerosis, hypertension, or other general causes (Ray and Stewart). Cases of lesser severity are amenable to medical treatment.

Physical Signs.—The tumours are ovoid or round in shape, situated at the bifurcation of the common carotid artery, lying partly under the sternomastoid, elevating the anterior border of that muscle, and partly in front of it. The surface is smooth or finely lobulated. The consistency is firm, resembling the texture of a fibrotic tuberculous gland. Lying within the carotid sheath, firmly attached to the artery, they can be moved from side to side but not vertically. On account of their vascularity, they can be temporarily reduced by pressure. Compression of the common carotid artery also temporarily reduces their size. Transmitted pulsation can always be elicited. A few tumours present a bruit and thrill. A sensitive carotid sinus, if present on the same side as the tumour, will give rise to the typical attacks on pressure on the tumour for about 30 seconds. The pharyngeal wall is occasionally displaced by the tumour and physical signs due to pressure on surrounding nerves are found occasionally.

Diagnosis.—The correct pre-operative diagnosis has only been made in 25 out of 180 tumours. The most common erroneous diagnoses are tuberculous glands, branchial cyst, aneurysm, or lateral aberrant thyroid tumour. Secondary malignant glands, branchial carcinoma, neurofibroma, lymphadenoma, and lymphosarcoma must also be considered. A punch biopsy has been suggested, but has been thought dangerous by most workers on account of the vascularity of the carotid body tumour. Even biopsy can be misleading, as reported by Walton (1946), where the section showed a typical carotid body tumour with islands of epithelioid cells separated by vascular stroma. A previous biopsy at another hospital and further investigation, however, showed this case to be a carcinoma of the arytenoid with secondary deposits in the cervical glands.

The criteria on which to base the diagnosis of carotid body tumours are :—

1. A history extending over several years of a slow-growing painless tumour in the neck.
2. A firm oval tumour in the region of the bifurcation of the common carotid.
3. Free lateral but no or very limited vertical mobility.
4. Decrease in size on compression of the common carotid artery.
5. Transmitted but not expansile pulsation.

Prognosis.—Prognosis depends on whether the common carotid has to be ligated and whether invasion has occurred before operation. The operative mortality is 15 per cent without ligation and 30 per cent when the artery has been tied.

Age and length of history are not a guide to whether malignancy has supervened. More rapid growth and horizontal fixation may be taken as an indication of malignancy. Usually it is only at operation when difficulty is encountered in shelling out that the invasive character of the tumour is manifest. Nor is age an infallible indication whether hemiplegia may occur after division of the common carotid. The youngest patient with a carotid body tumour in whom ligation of the common carotid resulted in hemiplegia was 26 years old. Pilcher and Tuss investigated 100 cases of hemiplegia after carotid ligation for various lesions. They did not find that age influenced the outcome of the operation.

Treatment.—Radiotherapy does not appear to have any effect on carotid body tumours. Phelps and Snyder (1937) reported 7 cases treated without benefit by irradiation. Stewart (1931) and Birman (1924) reported one case each which did not respond to X rays. Gordon-Taylor's case (1940), in which

radiation was tried and failed, turned out to be of nerve-cell origin. The case reported cured by X rays by Bevan and McCarthy never had a biopsy done and does not seem to have been adequately followed up.

The ideal treatment is total removal of the tumour without carotid ligation. Gordon-Taylor makes a plea for conservation of the common carotids and states that with careful dissection the carotids can be saved in a much larger number of cases than hitherto. Gratiot (1943) points out that up to 1920, 82 per cent were ligated, whilst in the years 1928–37 this figure fell to 50 per cent and he thinks that even this could be improved.

Hemiplegia may come on immediately after operation when it is due to deficient collateral circulation of the cerebral hemisphere or it may be delayed for 24 hours or longer when, as Keegan (1933) pointed out, it may be due to ascending thrombosis occluding the middle cerebral artery. The hemiplegia may be permanent, but often it is only transitory, leaving very little final disability, as in *Case* 2.

An electro-encephalogram after compression of the common carotid for 30 to 60 minutes is the best indication of the competence of the collateral circulation, although it does not exclude the danger of delayed hemiplegia (Lambert Rogers, 1944). If the electro-encephalographic studies show a deficient collateral circulation and during the course of the operation it becomes obvious that the tumour is inseparable from the vessels, it may be wiser to abandon the operation and to rely on what little help X rays can give.

In order to lessen the incidence of hemiplegia manual carotid compression for periods up to 30 minutes for two to three weeks before operation has been advised. The efficiency of the procedure has not yet been proved. This could be done by a series of E.E.G. studies. Mechanical devices to close the carotid gradually have been used : a clamp which can be tightened daily (Crile), metal bands (Halsted), and others.

Operation.—A general anæsthetic has been used in most cases, as the operation tends to be a long one. The incision should be generous and should either run along the skin creases or along the anterior border of the sternomastoid. The platysma is divided as well and the flaps raised to beyond the borders of the tumour. The deep fascia is incised along the anterior border of the sternomastoid and the muscle retracted posteriorly. The carotid sheath is now opened below the tumour, the common carotid isolated, and a ligature put around it, but not tied. This is a safeguard in case the artery is torn during the dissection. The internal jugular vein is either stretched across the tumour or pushed backwards and is usually divided. The vagus and sympathetic lie behind the tumour in the carotid sheath and must be carefully preserved. The superior laryngeal nerve of the vagus may be divided if it gets in the way. The hypoglossal nerve should be kept out of harm's way as it runs forward medial to the upper pole of the tumour. The tumour is freed from surrounding structures on all sides. It can be rotated from side to side to put all adherent structures on the stretch. An attempt should be made to dissect if off the arteries. If the vessels are not completely encircled this should be done by sharp dissection, but if they are completely surrounded Gratiot (1943) recommends inserting an artery forceps along the vessel from below and dividing the overlying tissues between clamps, much as one would separate the thyroid isthmus from the trachea. It is during this procedure that the vessel is most likely to be torn. If the vessel has to be tied it should be transfixed at the same time. The tumour can now be pulled upwards, exposing the vagus in its bed, and dissection proceeds from below upwards. The internal and external carotid arteries are then tied and transfixed and the tumour is removed. The skin and platysma are sutured, a small drain being left at the lower end of the wound.

Following ligation of the common carotid the patient should be kept in the Trendelenburg position for 10 days and not allowed up for another 2 to 3 weeks (Schorstein, 1940). This minimizes the danger of cerebral anæmia, which is the most common post-operative complication, giving rise to hemiplegia, aphasia, facial palsy, and unconsciousness. Deviation of the tongue would follow hypoglossal injury, and vocal cord paralysis would follow damage to the recurrent laryngeal nerve.

I wish to thank Mr. R. L. Newell for his advice and encouragement, and him and Mr. W. R. Douglas for permission to publish their cases, as well as Mr. F. Ward for the microphotographs.

BIBLIOGRAPHY

BEVAN, A. D., et al. (1929), *Surg. Gynec. Obstet.*, **49**, 764.
BIRMAN, A. (1924), *Dtsch. Z. Chir.*, **186**, 384.
BOYD, J. D. (1937), *Publ. Carneg. Instn.*, 479.
DE CASTRO, F. (1928), *Trab. Lab. Invest. biol. Univ. Madr.*, **25**, 331.
CHASE, W. H. (1933), *J. Path. Bact.*, **36**, 1.
DRIPPS, R. D., et al. (1944), *Amer. J. med. Sci.*, **208**, 681.
ENGEL, G. L., et al. (1944), *Arch. intern. Med.*, **74**, 100.
EWING, J. (1940), *Neoplastic Diseases*, 384.
FUNKE, L. (1904), *Amer. Med.*, **8**, 109.
GILFORD, H., et. al. (1904), *Practitioner*, **73**, 729.
GOODSITT, E., and SUDIMACK, G. (1945), *Amer. J. Surg.*, **67**, 131.
GORDON-TAYLOR, G. (1940), *Brit. J. Surg.*, **28**, 163.
GRATIOT, J. H. (1943), *Int. Abstr. Surg.*, **77**, 177.
HARRINGTON, S. W., et al. (1941), *Ann. Surg.*, **114**, 820.
KEEGAN, J. J. (1933), *Surg. Gynec. Obstet.*, **57**, 368.
KOHN, A. (1900), *Arch. micr. Anat.*, **56**, 81.
LUND, F. B. (1917), *J. Amer. Med. Ass.*, **69**, 348.
MARCHAND, F. (1891), *Festschr. R. Virchow*, **1**, 535.
MAURER, F. (1895), *Die Epidermis u. i. Abkömmlinge*, 72.
PALTOUF, R. (1892), *Beitr. path. Anat.*, **11**, 260.
PETERSON, E. W., et al. (1936), *Ann. Surg.*, **103**, 554.
PHELPS, F. W., et al. (1937), *West. J. Surg.*, **45**, 42.
PILCHER, C., et al. (1934), *Arch. Surg.*, **29**, 1024.
RANKIN, F. W., et al. (1931), *Ann. Surg.*, **93**, 801.
RAY, B. S., et al. (1942), *Surgery*, **11**, 915.
REID, M. R. (1920), *Bull. Johns Hopk. Hosp.*, **31**, 177.
ROGERS, LAMBERT (1944), *Brit. J. Surg.*, **32**, 309.
SCHORSTEIN, J. (1940), *Ibid.*, **28**, 50.
STEWART, H. H. (1931), *Ibid.*, **19**, 114.
DE TARNOVSKY, G. (1932), *Amer. J. Surg.*, **18**, 261.
WALTON, P. (1946), *Brit. J. Surg.*, **33**, 250.

THE SURGICAL TREATMENT OF HYDROCEPHALUS

A SIMPLE METHOD OF PERFORMING THIRD VENTRICULOSTOMY

By H. F. McNICKLE

WELLINGTON HOSPITAL, WELLINGTON, N.Z.

METHODS of performing third ventriculostomy which involve open craniotomy, such as those described by Dandy (1935) and by Stookey and Scarff (1936), seem rather severe in infancy, and are not free from unpleasant sequelæ. It is hoped, therefore, that the description of a simple and apparently safe method of performing third ventriculostomy (floor of the ventricle) will be of use to surgeons who, in the past, have hesitated to use more formidable procedures. Briefly, the method is to puncture the floor of the third ventricle by means of a hollow needle passed from above into a lateral ventricle, directed through the foramen of Monro into the third ventricle by touch or by telescope, and advanced to its destination under the X-ray screen.

In the past, third ventriculostomy has been reserved for cases of non-communicating hydrocephalus. The division of hydrocephalus into communicating and non-communicating types is an arbitrary one, and has been overemphasized. Two cases are reported to show that there are also types of communicating hydrocephalus which respond to third ventriculostomy.

This communication must be regarded as in the nature of a preliminary report, because the number of cases treated is small, and because no case has been followed for more than three years.

THE CAUSE OF HYDROCEPHALUS

Cerebrospinal fluid is formed by the choroid plexuses, leaves the ventricular system by the foramina of Luschka and of Magendie, to reach the subarachnoid space, where it is absorbed. Some fluid is absorbed in the spinal subarachnoid space, but most must pass forward through the basal cisterns and their branches to reach the cerebral subarachnoid space. Any obstruction in the main fluid pathways, inside or outside the ventricular system, results in hydrocephalus if the degree of obstruction is sufficient to interfere with the free flow of cerebrospinal fluid, and so cause the pressure in the ventricles (behind the obstruction) to be higher than the pressure in the cerebral subarachnoid space (beyond the obstruction).

The most complete obstructions are probably incompatible with survival for more than a few days. Fulminating hydrocephalus of this kind occasionally follows operation on a lumbar meningomyelocele. Hydrocephalus which pursues a more leisurely course is probably due to a partial obstruction, the degree of which is roughly proportional to the rate of progress of the disease.

The degree of obstruction does not always remain constant. Hydrocephalus sometimes progresses with increasing rapidity, as though the hydrocephalus, by distortion, were aggravating the primary obstruction. Probably a more common cause of swift deterioration is the development of a secondary obstruction, by compression of the main subarachnoid pathway at the level of the incisura tentorii (Cairns, 1939).

THE RATIONALE OF THIRD VENTRICULOSTOMY

If, then, hydrocephalus is due to an obstruction in the main fluid pathways, and such an obstruction can always be demonstrated at autopsy (Dandy, 1935), the rational treatment is to remove, or to by-pass, the block. In congenital hydrocephalus a direct attack upon the obstruction is seldom indicated. It is impossible in some cases, too hazardous in others, and in a third group direct intervention is followed by recurrence. Ventriculostomy is simply an attempt to by-pass the obstruction. As Dandy (1935) pointed out, the ideal spot for draining the ventricles into a cistern is just behind the pituitary stalk, in the floor of the third ventricle. An opening made here, even a small one, has a good chance of being permanent, because of the thinness of the floor, relative absence of neuroglia, and the depth of the cistern beneath.

Cauterization of the choroid plexus, as practised by Scarff (1936), Dandy (1938), Sachs (1942), and Putnam (1943), seems to be wrong in principle. As long as the block remains, the pressure in the ventricle will be higher than the pressure in the cerebral subarachnoid space, and the ventricles will continue to expand. A reduction in the volume of cerebrospinal fluid formed may retard the rate of expansion, but seems unlikely to effect a complete cure. Surely, too, apart from the immediate risk of operation, there is the considerable danger that floating particles from the operation will be carried to the site of obstruction, will lodge there, and will cause an acute, perhaps fatal, exacerbation of the condition.

HYDROCEPHALUS ASSOCIATED WITH LUMBAR MENINGOMYELOCELE

The regular association of the Arnold-Chiari malformation with lumbar meningomyelocele was pointed out by Russell and Donald (1935). Hydrocephalus when it occurs (it frequently follows operation on the lumbar sac), is usually communicating in type, the block being in the subarachnoid space around the brain-stem at the foramen magnum. The probable explanation is that the spinal cord, firmly fixed in the lumbar region, is elongated by the growth of the spinal column, and so draws part of the brain, including the foramina of Luschka and of Magendie, into the spinal canal. In a severe case brain elements may be found as low as the seventh cervical vertebra. Operation on the lumbar region may precipitate matters by the sudden release of cerebrospinal fluid or by traction on nervous elements attached to the sac. All cases must be regarded as being potentially hydrocephalic.

Many cases of meningomyelocele should not be subjected to operation. If there is no hydrocephalus, no increase in the size of the sac, and no immediate danger of ulceration or rupture of the sac, they are best left alone. Many cases are too severe. For example, it is hardly justifiable to operate when there is complete paralysis of lower limbs and of sphincters. No improvement in the paralysis can be expected to follow operation.

For cases in which operation is contemplated, the following programme is suggested :—

1. If hydrocephalus is already present it should be treated first, by third ventriculostomy.
2. Before opening the lumbar sac, pressure should be released from above, by tapping a lateral ventricle.
3. If a general anæsthetic is used a perfect airway must be maintained.
4. The table should be tilted steeply, head down.
5. There must be no traction on cord or coverings.
6. If hydrocephalus develops after operation in spite of these precautions, third ventriculostomy should be performed.

D'Errico (1938) has performed an extensive cervical laminectomy for the relief of hydrocephalus in these cases, with some success. Third ventriculostomy appears to be simpler and safer (*Case* 2, *Fig.* 451), though a bone operation may be indicated later for the relief of other symptoms.

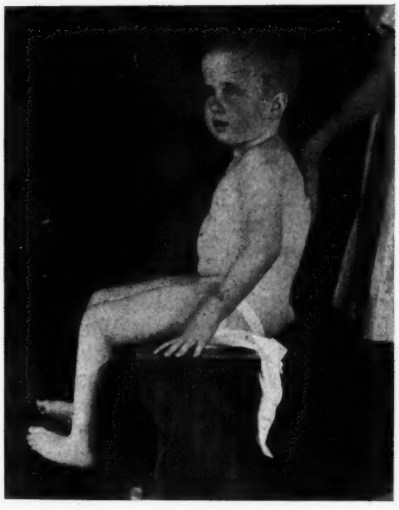


FIG. 451.—*Case* 2. Sacral meningomyelocele causing paralysis of sacral nerves, and associated with hydrocephalus of mild degree. The hydrocephalus responded to third ventriculostomy.

DIAGNOSIS OF CONGENITAL HYDROCEPHALUS

The advanced case may be diagnosed by inspection, but a certain diagnosis cannot be made in the early case merely on the grounds of enlargement of the head, with large, tense, or bulging fontanelle. A number of such cases will be found on ventricular puncture and ventriculography to have no enlargement of the ventricles. Some alleged spontaneous cures may be in cases of this type. In all doubtful cases, and these will include the majority if an early diagnosis is to be made, the lateral ventricle should be punctured through the anterior fontanelle. The scalp is punctured obliquely, and a fine-bore needle is used to minimize the risk of subsequent leakage. Usually the cortex is thinned to a remarkable degree, and the ventricle is entered soon after puncturing the dura. If the diagnosis is still in doubt, a volume of fluid is withdrawn from the ventricle, a considerably smaller volume of air is injected, the exchange being made gradually, and radiographs are taken.

INVESTIGATION

It has been customary to divide hydrocephalus into communicating and non-communicating types. In the first there is free communication between the ventricles and the spinal subarachnoid space. In the second there is not. Whatever tests are used care should be taken not to make unwarranted assumptions from them.

Phenolsulphonephthalein injection, recommended by Dandy (1935), has proved satisfactory in our hands. Our procedure is as follows :—

1. Preliminary tap of a lateral ventricle to relieve the pressure above the obstruction. Possibly some ill effects attributed by others to phenolsulphonephthalein are due to neglect of this precaution.
2. Lumbar puncture is performed, and ½ c.c. of phenolsulphonephthalein solution is injected. More than ½ c.c. may be used if the head is large.
3. After 20 minutes the ventricle is tapped again and the colour of the fluid is noted.

a. An intense colour means that there is free communication between the ventricular system and the spinal subarachnoid space (communicating type) The obstruction is in the fluid pathways outside the ventricular system ; but this does not mean that the interpeduncular cistern beneath the floor of the third ventricle is obliterated. The block will frequently be behind this point, and so relief will follow third ventriculostomy. There is no way to determine the patency of the interpeduncular cistern prior to operation.

b. If no dye has reached the ventricle in 20 minutes the hydrocephalus is said to be non-communicating. Relief should follow third ventriculostomy.

4. In non-communicating cases Dandy (1935) recommended that the rate of dye excretion in the urine be measured. The urine collected in two hours is alkalinized if necessary, and the volume compared with the volume of ½ c.c. of dye diluted to match. If the quantity of dye appearing in the urine approaches 40 per cent of the quantity injected, there must be free communication between spinal and cerebral subarachnoid spaces and a normal rate of absorption.

Dandy thought that the excretion of less than 10 per cent of the dye indicated an obstruction of the subarachnoid pathways in addition to a ventricular block, and was a contra-indication to third ventriculostomy. However, this subarachnoid component, when it exists, is probably only secondary. Hydrocephalus may cause a secondary compression of the subarachnoid pathways at the incisura tentorii or at the foramen magnum. If the primary obstruction can be short-circuited, one would expect the secondary component to be relieved also. The dual nature of the obstruction in some cases of hydrocephalus

has been emphasized by Cairns (1939). Two of our non-communicating cases showed a poor dye excretion, in one case due to a low output of urine. Both showed a satisfactory dye excretion when the test was repeated, and both responded to third ventriculostomy.

INDICATIONS FOR THIRD VENTRICULOSTOMY

1. Congenital hydrocephalus. Whatever the type of hydrocephalus, if any treatment is to be carried out, third ventriculostomy by the method to be described appears to be the treatment of choice. It may fail to relieve some cases of communicating hydrocephalus, but this by itself is no bar to the use of a procedure attended by so little risk.

There is no point in operating on the advanced case (although it is remarkable how long the faculties are preserved) or on the case with evidence of other intracranial defect. If operation is to be performed, the sooner it is done the better. If for any reason operation is delayed, the head should be kept at a reasonable tension by ventricular taps. Any large quantity of fluid thus obtained is best re-injected subcutaneously to prevent dehydration and demineralization.

2. Acquired hydrocephalus. Both post-meningitic hydrocephalus and benign stricture of the aqueduct of Sylvius appear to be suitable for third ventriculostomy. No case of the latter has yet been treated by us. Space-occupying lesions causing hydrocephalus are usually subjected to direct attack. However, when a tumour is inoperable, third ventriculostomy may sometimes be considered as a palliative.

THE OPERATION

1. Preliminaries.—

a. A burr hole must be made in older patients, but this step is unnecessary in infants when the site of needle puncture falls within the anterior fontanelle.

b. Enough fluid is removed from a lateral ventricle to reduce the tension to a reasonable level. This reduces leakage at operation, and tends to deepen the interpeduncular cistern. If the case is one of non-communicating hydrocephalus, 10 or 15 c.c. of the ventricular fluid is mixed with ½ c.c. phenolsulphonephthalein, and is reinjected by lumbar puncture. This procedure will further deepen the interpeduncular cistern, and will colour the fluid it contains.

c. The patient is transferred to the X-ray screening room, where apparatus has been assembled for both postero-anterior and lateral screening. Metal markers are attached to the skin in the midline over the glabella and over the inion, and on either side above and in front of the ear, and as close as possible to it. The midline markers must be placed with great accuracy. If there is any doubt about the position of the inion, or if there is asymmetry of the head, a radiograph to show the foramen magnum is taken as a check. Patient and X-ray equipment are then arranged as for operation, and a lateral radiograph is taken to show the position of the sella turcica in relation to the lateral markers (*Fig.* 452). The markers need not be accurately superimposed on the film, but their position on the film must be exactly reproduced on the fluorescent screen at operation.

2. Anæsthetic.—For infants a general (non-inflammable) anæsthetic is the most reliable. Our preference is for a little trilene administered on an open mask. For older patients local anæsthetic is used.

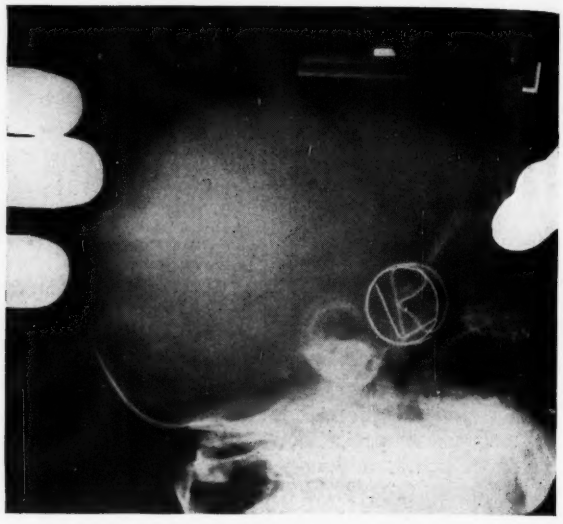


FIG. 452.—Lateral radiograph taken immediately before operation. At operation, after the needle has entered the third ventricle, the radiologist uses these markers as a guide to the sella turcica.

3. Instruments.—A 19-gauge hollow needle of sufficient length. The point must be ground right off and the end rounded; the stylet must fit loosely so that it may be removed from the needle without a jerk.

4. Present Method.—

a. A tenotome puncture is made through scalp and dura, preferably on the right side, ½–1 in. from the midline, depending on the size of the head, and ½–1 in. behind the coronal plane which passes through the temporomandibular joints. The hollow needle is passed through this puncture and on into the lateral ventricle. The needle must not be gripped or hindered in any way by scalp or dura.

b. The needle is advanced towards the floor of the anterior horn a centimetre or two in front of the foramen of Monro by directing the needle slightly towards the midline and a little in front of a line joining the temporomandibular joints. As the floor of the ventricle is approached, the needle is halted once or twice, and the needle point is moved from side to side to touch the side-walls of the ventricle. By this manœuvre the side-walls are easily avoided, and the needle point is carried safely to the floor of the ventricle.

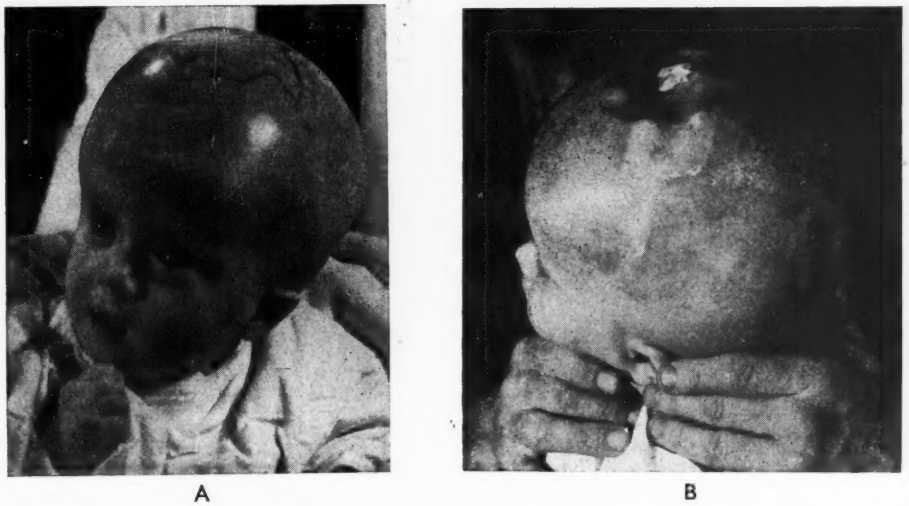
c. The operator then feels his way posteriorly, tapping along the floor of the anterior horn, like a blind man with his stick, until the needle falls into the third ventricle. The lining of the lateral ventricle is resistant enough to a blunt needle for this to be done quite easily and safely.

d. The needle is moved gently from side to side to feel the sides of the foramen of Monro; then, with the needle centred in the foramen, the point is

advanced to puncture the floor of the third ventricle. The operator takes both hands from the needle and steps back.

e. The radiologist screens the head postero-anteriorly and laterally. In determining the position of the needle he relies on the metal markers. During lateral screening the head must be moved until the lateral markers occupy exactly the same position on the fluorescent screen as they did on the lateral film. Usually the needle is found to have reached the right spot or to be within a millimetre or two of it. If the radiologist thinks that the position of the needle can be improved, lights are turned on while the operator withdraws the point into the third ventricle, and punctures the floor again. Screening is repeated to determine the new position. Rarely will a third puncture be found necessary.

f. In non-communicating cases the stylet is withdrawn. A flow of coloured fluid confirms that the point lies in the interpeduncular cistern.



A B

FIG. 453.—*Case* 1. A, Anterior fontanelle outlined the day before operation. B, 2 days after operation. There is extreme depression of the fontanelle. 110 c.c. of fluid was injected into a lateral ventricle without completely relieving the depression. A small dressing shows where the needle was introduced at operation.

g. The needle is removed. The point is not agitated before withdrawal. Simple puncture probably provides an adequate and permanent opening. Any wide movement of the needle point is dangerous.

h. A suture is placed in the scalp to prevent leakage and a collodion dressing is applied.

EARLIER METHOD

In the first four cases treated the needle was passed through the foramen of Monro under vision. A telescope was introduced into the lateral ventricle through a separate puncture placed far enough lateral to the needle puncture to allow the two instruments to be manipulated independently. The foramen of Monro was at once identified by the choroid plexus, which enters the foramen from behind; the needle was passed through the foramen, and the telescope was withdrawn. Subsequent steps were the same as those given above.

Comment.—The only disadvantage of the earlier method was that two punctures of the cortex had to be made instead of one. The telescope helped us to become familiar with the interior of the hydrocephalic lateral ventricle, but, in the end, proved to be unnecessary.

When the foramen of Monro is large (in a boy of 14 years it appeared to be enormous), it is possible to pass a telescope right into the third ventricle as Mixter (1923) did. We have not attempted to do this in the living. We feel that the methods described above are easier, safer, and more generally applicable.

POST-OPERATIVE COURSE

Early crises have sometimes followed other types of operation and have been attributed by some to the sudden reduction of intracranial pressure. We have not observed any post-operative upset of any kind. The usual course of events in infants is that the bones of the vault fall together and engage during the first 24 hours; thereafter the anterior fontanelle, now reduced in size, becomes deeply concave during the next two or three days (*Fig.* 453), then the concavity slowly disappears, and is gone by the end of the first week. Possibly the choroid plexuses take some days to recover from the effects of prolonged intraventricular pressure, and to reach their normal secreting capacity. Extreme depression of the fontanelle should be relieved, to protect cerebral blood-vessels, by injecting fluid into a lateral ventricle (*Case* 1). Hartmann's solution is suitable for this purpose.

The escape of fluid from ventricle to subarachnoid space through the punctures made in the cortex may be discounted as a factor in producing improvement, because no benefit has ever followed simple punctures of the cortex up to 3 mm. diameter (telescope size), although such punctures of a thinned-out cortex remain patent for many weeks.

Puncture of the floor of the third ventricle might be expected to produce some disturbance of the autonomic nervous system. In practice this has not occurred. There has been no unpleasant sequel of any kind.

RESULTS

NON-COMMUNICATING HYDROCEPHALUS

Four cases have been treated. All were markedly relieved by operation. The first case treated was a female infant with a severe degree of obstruction (*Fig.* 454). She has now been followed for three years. Development, behaviour, and intelligence all appear to be normal. The circumference of the head has increased by less than ¼ in. in the last six months.

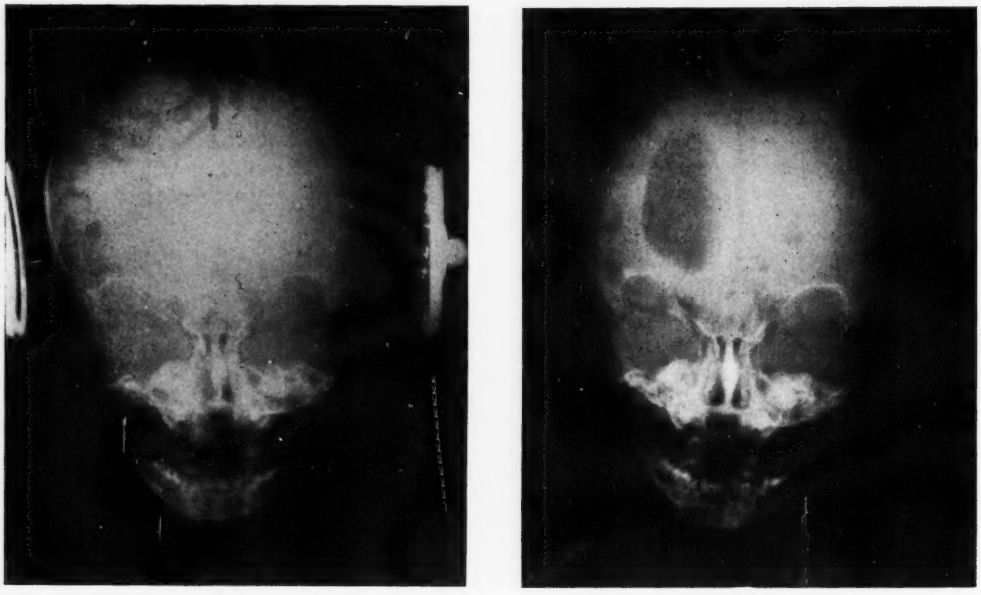
Two other infants with severe hydrocephalus have been treated. One died of an unrelated acute respiratory infection two months after operation.

CASE REPORTS

Case 1.—C. K., female aged 12 months, with communicating hydrocephalus of moderate severity.

HISTORY.—Enlargement of the head was noticed at 2 months. For the last six months she had been in an institution on a diet of cow's milk. No solid food had been added. She was generally retarded, but recently she had shown some interest in toys, and had attempted to raise herself by grasping the sides of the cot.

ON EXAMINATION.—She was apathetic and overweight. Two teeth had erupted. The circumference of the head was 22 in. The shape of the head and the outline of the anterior fontanelle are shown in *Fig.* 453. It was thought her backwardness was partly due to diet and environment.



A B

FIG. 454.—Non-communicating hydrocephalus. A, Encephalogram before operation, with head right side up, showing air in the subarachnoid space, none in the ventricles. B, Encephalogram one month after operation, with head right side up. Air freely enters the ventricular system. This case has been followed three years.

The other, now followed for nine months, is progressing most satisfactorily.

The fourth case, a boy of 14 years, said to have had meningitis at the age of 2 years, was treated because of increasing spasticity. It was feared that he would soon become bedridden unless something was done. The head circumference was 27 in. Symptomatic improvement after operation was beyond expectation. Twitching and vomiting ceased; spasticity was greatly improved. There has been no relapse in one year.

COMMUNICATING HYDROCEPHALUS

Three cases have been treated. The first case was unrelieved. It is possible that failure in this case was due to a technical error. The puncture may have been made away from the midline because of gross asymmetry of the head. Unfortunately there was no radiographic check of the position of the midline markers, nor was the operation repeated. There was no autopsy.

The other two cases, one due to the Arnold-Chiari malformation, were treated recently. Nevertheless, they seem to be worth reporting, because it has been widely assumed that communicating hydrocephalus will not respond to third ventriculostomy.

OPERATION (Nov. 24, 1945).—No telescope was used. A small dressing shows where the needle was introduced (*Fig.* 453, B). Two days later, depression of the anterior fontanelle was extreme (*Fig.* 453, B). The scalp was sharply drawn in over the fontanelle margins. The floor of the depression was tight and rigid; it did not pulsate, and it moved only slightly when the infant cried. There were no symptoms apart from slight drowsiness, but it was felt that thin-walled blood-vessels might give way if the intracranial pressure were not raised. Accordingly, 110 c.c. of Tyrode's solution (Hartmann's solution was not available) was allowed to run into a lateral ventricle. Even this did not entirely relieve the depression of the fontanelle. During the next two days the fontanelle was again deeply indrawn, but not quite to the same extent. No more fluid was injected. The concavity had disappeared by the eighth day. Now, after five weeks, the infant is happy, bright, and responsive. Part of the improvement is due, of course, to a change of diet and to more personal attention.

Case 2.—M. T., female aged 7 years. Mild degree of hydrocephalus, associated with sacral meningomyelocele (*Fig.* 451).

HISTORY.—She was admitted to an institution when she was a few weeks old. The head has slowly increased in size, but the sacral sac and the paralysis of legs and sphincters, so far as anyone knew, had not altered. She had never been out of bed.

ON EXAMINATION.—She was a friendly, happy, and attractive child of normal development and nutrition. She was mentally retarded, but less so than one would expect as a result of her confined existence. The head circumference was 24¼ in.; but, apart from enlargement of the head, hydrocephalus appeared to cause no symptom, nor was there any evidence of brain-stem compression. The upper limbs were normal. Over the upper part of the sacrum was a tough, thick-walled sac, which measured 2½ in. in diameter. There were bed-sores 2 in. in diameter over the ischial tuberosities. Structures supplied by sacral nerves showed almost complete paralysis; but the thigh muscles were strong, and easily able to support the body-weight. There was a little fixed flexion of hips and knees. It was thought that she could be made to walk, and that orthopædic treatment would be well worth while if the hydrocephalus could be arrested. There was no indication to interfere with the sac in the sacral region.

OPERATION (Dec. 1, 1945).—Operation was performed under local anæsthetic, without using a telescope. In the days following operation, changes in cerebrospinal fluid pressure, although less marked than in *Case* 1, were clearly reflected in the size and tension of the sacral sac, which became smaller, softer, and more wrinkled. Changes in size were recorded by taking plaster casts of the sac. During the month since operation her vocabulary and general knowledge have increased enormously; and she has learned to walk with the aid of a walking frame.

SUMMARY

1. A simple and apparently safe method of performing third ventriculostomy (floor of the ventricle) by means of a hollow needle introduced from above by way of the lateral ventricle, foramen of Monro, and third ventricle is described.

2. It is hoped that the method will be of use to surgeons who, in the past, have hesitated to use more formidable procedures.

3. In the past, third ventriculostomy has been reserved for cases of non-communicating hydrocephalus.

4. The division of hydrocephalus into communicating and non-communicating types is an arbitrary one, and has been over-emphasized. Two cases are reported to show that there are also types of communicating hydrocephalus which respond to third ventriculostomy.

5. Cauterization of the choroid plexus seems to be wrong in principle. Also floating debris after operation must sometimes aggravate the hydrocephalus.

6. Suggestions are made for the prevention and treatment of the type of hydrocephalus associated with lumbar meningomyelocele.

I am greatly indebted to Dr. N. F. C. Hill for all the radiological work; and to Dr. Murray A. Falconer for helpful advice in preparing this paper.

REFERENCES

CAIRNS, H. (1939), *Brit. J. Surg.*, **27**, 275.
DANDY, W. E. (1935), *Practice of Surgery* (Lewis, D.) **12**, I, s. 5. Hagerstown, Md.: W. F. Prior Co. Inc.
— — (1938), *Ann. Surg.*, **108**, 194.
D'ERRICO, A. (1938), *Surgery*, **4**, 856.
MIXTER, J. (1923), *Boston med. surg. J.*, **188**, 277.
PUTNAM, T. J. (1943), *Surg. Gynec. Obstet.*, **76**, 171.
RUSSELL, D. S., and DONALD, C. (1935), *Brain*, **58**, 203.
SACHS, E. (1942), *J. Mt. Sinai Hosp.*, **9**, 767.
SCARFF, J. E. (1936), *Arch. Neurol. Psychiat.*, **35**, 853.
STOOKEY, B., and SCARFF, J. E. (1936), *Bull. neurol. Inst. N.Y.*, **5**, 348.

CEREBRAL THROMBO-ANGIITIS OBLITERANS

BY LOYAL DAVIS AND GEORGE PERRET

FROM THE DEPARTMENT OF SURGERY, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, CHICAGO

UNTIL recent years thrombo-angiitis obliterans was known as a disease affecting primarily the peripheral blood-vessels of the extremities. From the publications of Barron and Linenthal (1929), Perla (1925), Cserna (1926), and others, and especially the examination of autopsy material by Jaeger (1932), it became known that the vessels of the heart, kidneys, stomach, and pancreas are often similarly affected. It was found that the disease might start in visceral vessels and not involve the periphery at all, or that it might affect the peripheral and central vessels simultaneously. Gresser (1932) and Marchesani (1934) described a similar process in the vessels of the retina.

Cerebral symptoms associated with peripheral thrombo-angiitis obliterans have been reported occasionally in the literature during the past ten years. In a study of 500 cases of peripheral thrombo-angiitis obliterans Hausner and Allen (1938) found the evidence of a cerebral lesion in 11, or 2 per cent, of the cases.

The purely cerebral form as first observed and described by Foerster and Guttmann (1933) and studied anatomically by Spatz (1935) and later by Bielschowski (1936), Stender (1936), Benedek (1936) Lindenberg (1939), Rosenhagen (1939), Antoni (1941), and others, presents many diagnostic difficulties. It has been diagnosed frequently as lues, juvenile arteriosclerosis, hypertension, atypical multiple sclerosis, or an intracranial tumour. The condition is not as rare as might be expected and may very well be responsible for many organic cerebral changes which are not clearly diagnosed and which masquerade under the name of other diseases. Symptoms of increased intracranial pressure are rarely present and often the symptoms are only those of a localized cortical process. In many instances intermittent claudication follows the initial cerebral symptoms and may aid in making an accurate diagnosis. In the cases of true cerebral thrombo-angiitis obliterans reported in the literature a diagnosis of cortical atrophy due to a vascular lesion was made in a few instances following encephalography or ventriculography. The air studies showed a unilateral or bilateral enlargement of the lateral ventricles and enlarged subarachnoidal spaces over one or both hemispheres. Obliterated cerebral arteries have been described by Sorgo (1939) and

Sunder-Plassmann (1941) in arteriograms of patients with Buerger's disease. However, the majority of the reported cases were diagnosed at autopsy.

Cerebral thrombo-angiitis obliterans occurs most commonly in people between the ages of 30 and 50 years. The course of the disease is chronic and progressive, but frequently presents sudden attacks of temporary symptoms and complete remissions.

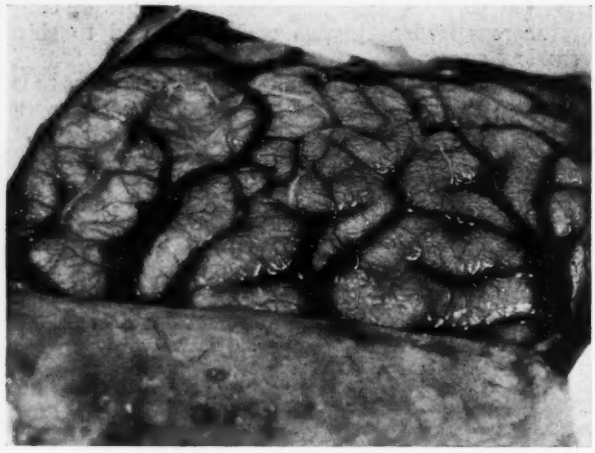


FIG. 455.—Operative exposure of the right fronto-parietal region (*Case* 1). The thin, white, string-like, obliterated vessels are seen branching off from normal larger vessels. The cortex below the obliterated vessels is pale, yellow, and atrophic.

The symptoms depend upon the location and the extension of the diseased vessels and for this reason they can be numerous and widespread. The most common are headaches, paræsthesias, pareses, aphasia, and visual defects. Convulsive seizures may be observed and psychic and memory changes are frequently present. In the following 11 cases a diagnosis of a localized cerebral lesion was made. The symptoms were those of an intracranial space-occupying lesion but it was not possible to rule out a vascular lesion. The clinical pictures pointed to definite localizations and the severity and progression of the symptoms justified a craniotomy in 9 of the cases.

CASE REPORTS

Case 1.—A 34-year-old female had a history of gradual onset of clumsiness and paræsthesia in the left hand following a fall while ski-ing. After four weeks' duration this condition gradually disappeared, and she was free of symptoms for ten months. She then suddenly developed a paralysis of the left leg which was followed by a convulsive seizure involving the entire left side of the body. Paræsthesias, weakness, and clumsiness of the left upper extremity soon developed. The neurological examination performed a few days later revealed a slight left facial weakness and a paresis of the left upper extremity most marked in the hand. The paralysis of the left leg had entirely disappeared. Subjective hyperæsthesia of the left upper extremity, impairment of joint and vibratory sense in the fingers of the left hand, a marked loss of stereognosis in the left hand as well as a marked clumsiness were present. Rapidly alternating movements, finger-to-nose and finger-to-finger tests were poorly done with the left hand, and all the deep reflexes were hyperactive on the left side. Her blood-pressure was 120/70 and her serology negative. A diagnosis of a right fronto-parietal lesion was made, and at operation an extensive yellowish and atrophic area was found posterior to the central sulcus, through which ran obliterated cortical vessels appearing like thick white threads (*Fig.* 455). The patient was put on bromide therapy and had no convulsions until 20 months later, four weeks before the normal delivery of a healthy baby. She has been well since (18 months), although she has tempory periods of depression.

Case 2.—A 38-year-old male gave a history of numerous attacks of temporary paræsthesia, clumsiness, and paresis of the left extremities and the left side of the face over a period of eight years. More frequent and severe attacks associated with increased weakness of the left arm and hand, speech disturbances, memory defects, and inability to concentrate had occurred in the four weeks immediately preceding hospital entrance. At examination there was a marked muscular weakness of the left forearm, hand, and fingers involving especially the flexor groups. An objective hyperæsthesia of the distal phalanges as well as a subjective hyperæsthesia over the entire left upper extremity and the left side of the face was present. Stereognosis was slightly diminished in the left hand. The finger-to-finger and finger-to-nose tests and rapidly alternating movements were poorly performed with the left hand. All of the deep reflexes were hyperactive on the left side. Words were clearly but slowly spoken and the patient stated that he had to picture the written words in his mind before pronouncing them. His memory for recent events was poor and he had difficulty with simple arithmetic problems. He often cried and laughed out of proportion to the stimulus and usually without reason. His blood-pressure was 120/80 and the serology negative. A diagnosis of a right temporo-parietal lesion was made, and at operation a thickened arachnoid and numerous obliterated cortical vessels were found over the right parietal and temporal lobes with neighbouring areas of atrophy and softening (*Fig.* 456). Three and a half years after the operation he has occasional attacks of tingling in the left arm, hand, and foot and is facetious, but is working regularly.

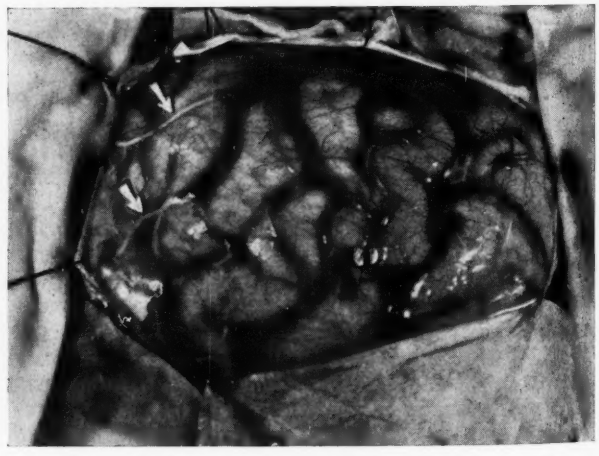


FIG. 456.—Operative exposure of the right fronto-parietal region (*Case* 2), showing disseminated white and obliterated vessels over granular appearing cortex.

Case 3.—A 47-year-old male gave a one-year history of severe and progressive paræsthesias in both his right extremities. During the three months before examination he developed a right hemiparesis and transitory speech disturbances. At examination the movements of the right hand were slow and awkward and alternate movements were performed clumsily with the right fingers and hand. The deep reflexes were all hyperactive on the right side. His speech was slow and measured

and he had difficulty in enunciating some of his words. At the time of examination the paresis of the right leg had receded almost completely. His blood-pressure was 150/80 and the blood serology negative. A diagnosis of a tumour of the left parietal lobe was made and an osteoplastic craniotomy performed. Over the motor and post-central regions numerous small, disseminated areas of yellowish degenerated and atrophied cortex were found, through which ran thin, white, completely obliterated vessels. Over the neighbouring normal-appearing areas the small cortical vessels were greatly increased in number and the arachnoid was thickened. The patient died 17 days following the operation of a bronchopneumonia of the right lung. At autopsy, sections of the brain and superficial cerebral vessels revealed inflammatory changes in the vessel walls. Few polymorphonuclears but many lymphocytes and a large number of young fibroblasts were present in the intima and the subintimal layers of the vessels. The lumen of the vessels was completely filled with large organizing thrombi. This picture was found in sections through vessels in various areas of the cerebral cortex. Engorgement of the capillaries, inflammatory changes around their wall, and glial proliferation were also present.

Case 4.—A 53-year-old male gave a history of pain and weakness in the lower part of his back for eight years. During the 15 months prior to his hospital admission he developed a paraplegia which was followed by paralysis of the left upper extremity and urinary incontinence. At examination he was unable to sit or walk, the left upper extremity was completely paralysed, and both lower extremities showed a spastic paresis which was more marked on the left. Superficial and deep sensation was critically diminished over the entire left side and only slightly over the right lower extremity. All of the deep reflexes were hyperactive in the lower extremities and pathological plantar reflexes were present. The deep reflexes of the left upper extremity were also hyperactive. There were a left homonymous hemianopsia, a moderate degree of dysarthria, and sphincter disturbances. The patient was disorientated and showed numerous mental and psychic changes. His blood-pressure was 170/110 and the serology negative. A diagnosis of an intracranial lesion in the parasagittal area was made and a right osteoplastic craniotomy performed. When the dural flap was raised the entire exposed area showed a yellowish, soft, degenerated cortex with white obliterated vessels running in the deepened sulci. The patient died 36 days after the operation.

Case 5.—A 62-year-old man had gradually progressing symptoms of weakness and spasticity in the right upper extremity and speech difficulties. At examination his speech was slow and hesitant and he had some trouble in finding words. When reading, words were skipped and others put together. He presented a right facial weakness and weakness, clumsiness, and astereognosis in the right hand. He had no objective disturbances of superficial sensations. The deep reflexes were more active in the right than in the left upper extremity. The blood-pressure was 160/80 and the serology negative. A diagnosis of a left fronto-parietal tumour was made and an osteoplastic craniotomy performed. The arachnoid was thick, opaque, and distended with fluid, and cortical atrophy was present. The cortex was yellow and atrophic in the precentral region and in the surrounding gyri the number of the cortical vessels was increased as if there had been an attempt to establish a collateral circulation. Over the atrophied areas several thin, white, and string-like obliterated arteries were present (*Fig.* 457). Following the operation, the patient developed convulsive seizures which were controlled by bromide therapy. One year later the patient had some weakness in the right arm and leg and was emotionally disturbed by his progressive speech defect.

Case 6.—A 37-year-old woman developed six months before her admission in the hospital a sudden transitory loss of vision and later a slowly progressive left visual defect. She suffered from frequent headaches and then developed paræsthesias and weakness in the left upper extremity. Examination revealed a left homonymous hemianopsia, a weakness in the left side of the face, and increased deep reflexes in the left extremities. Ventriculography showed the ventricles to be within normal limits.

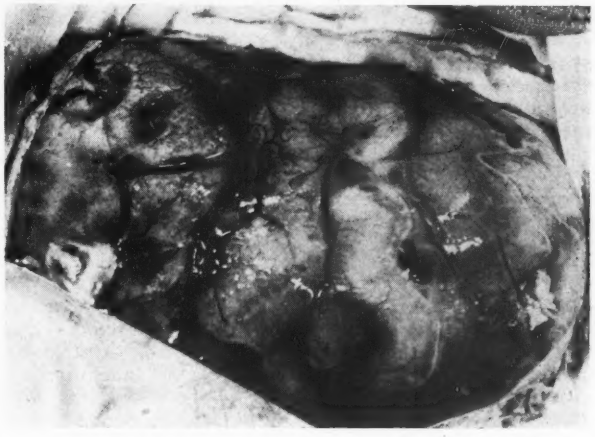


FIG. 457.—Operative exposure of the left fronto-parietal region (*Case* 5), showing disseminated white and obliterated vessels, areas of cortical atrophy, and evidence of old perivascular inflammation involving the arachnoid.

Following this procedure she developed a transitory right homonymous hemianopsia. Her blood-pressure was 125/85 and the serology was negative. A right temporal lesion was diagnosed. An osteoplastic craniotomy was performed and a thickened arachnoid, obliterated cortical vessels, and atrophied gyri found. Nine months after the operation the patient still had the left homonymous hemianopsia and occasional attacks of paræsthesia in both hands.

Case 7.—A 42-year-old woman had a four-year history of temporary diplopia, blurring of vision, and reading difficulty. Two years previous to hospital admission she developed sudden paræsthesias in the left arm and shoulder and was completely aphasic for a few hours. She also had personality difficulties and domestic problems which she was not interested in solving. One year later she developed recurrent temporary periods of numbness over the left side of the head, face, neck, and left upper extremity, with speech difficulties. One month prior to her admission in the hospital she had severe headaches, associated with nausea and marked reading difficulties. On examination she had a right incomplete homonymous hemianopsia, a complete alexia but could spell out individual letters, hesitancy in speech, and some weakness in the left upper extremity. The skin of all the extremities appeared mottled and was usually cold. The ulnar, radial, posterior tibial, and dorsalis pedis pulsations were palpable. The blood-pressure was 160/95 and the serology negative. Oscillometric readings were diminished in both feet and hands, but about equally. Immersion of the hands in warm water diminished the skin temperature in both lower extremities and produced pain in both hands and arms. Ventriculography disclosed equally dilated ventricles with large subarachnoid spaces and evidence of bilateral cortical atrophy. A pre-operative diagnosis of thrombo-angiitis obliterans was made in view of the transient and bilateral symptoms and the ventriculographic findings. An osteoplastic craniotomy revealed a thickened arachnoid, a large subarachnoid space with atrophic gyri, and many small,

white, pipe-stem, completely obliterated arteries scattered over the frontal and parietal cortex. She was sent to a dry, warm climate and her condition had somewhat improved six months after the operation.

Case 8.—A 50-year-old woman presented a seven-year history of nocturnal, convulsive seizures, associated in recent months with headaches, visual disturbances, and cold extremities. On examination she showed some clumsiness in the left hand and several thin white obliterated vessels could be observed on the left retina with signs of periangiitis. Her blood-pressure was 150/90 and the serology negative. Encephalographic studies revealed symmetrically dilated ventricles with enlarged subarachnoid spaces, especially in both frontal areas. A diagnosis of cerebral thrombo-angiitis obliterans was made and the patient put on anticonvulsant therapy. She later had an episode of marked mental disturbances which cleared up completely. She is working and complains of cold extremities and has an occasional nocturnal convulsion.

Case 9.—A 63-year-old man had developed at the age of 62 transient paræsthesias in the left 4th and 5th fingers with progressive inability in performing movements with the left hand, progressive weakness and numbness in the entire left upper extremity, and later clumsiness in the left foot and weakness in the entire left lower extremity. Examination revealed a left homonymous hemianopsia, weakness and ataxia in the left upper and lower extremities, and hyperæsthesia in the left upper extremity. The finger-to-finger and finger-to-nose tests were poorly performed with the left hand. There was no loss of vibratory or joint sensation, but he had an astereognosis in the left hand. The deep reflexes were hyperactive on the left side and he had a left positive Babinski sign. The blood-pressure was 140/80 and the serology negative. A diagnosis of a right temporal neoplasm was made and at operation large subarachnoid spaces were found with areas of granular atrophy of the cortex and the presence of numerous white, obliterated vessels. The patient developed a bronchopneumonia and died on the fifth post-operative day. Unfortunately, an autopsy was not granted.

Case 10.—A 52-year-old Jewish man gave a three-weeks' history of paræsthesias in the entire left lower extremity with attacks of anæsthesia accompanied by convulsive movements. He also had occasional paræsthesia in the left upper extremity. Examination revealed no muscular weakness or sensory disturbances. The deep reflexes were equal and there were no pathological reflexes. His blood-pressure was 120/86 and the blood serology was negative. Good pulsations were felt in all palpable arteries. Plethysmographic and oscillometric studies failed to show any abnormalities or differences between the right and left extremities. Skin temperature studies were within normal limits and showed a definite rise following immersion of the hands in hot water, but no drop following immersion of the hands in ice water. Ventriculography was performed and no deformity, defect, or displacement of the ventricular system significant of a space-occupying lesion could be seen, but it was not conclusive evidence. At operation the arachnoid was found distended with fluid, especially over the motor and premotor areas; it was also somewhat thickened, especially along the course of the vessels. The gyrus immediately posterior to the Rolandic vein and near the sagittal sinus was yellow and showed evidence of granular atrophy. Small, white, obliterated arteries were present in that area, together with a thrombosed vein. The patient was put on an anticonvulsant therapy. Two months after discharge from the hospital the patient was well and had had no recurrence of his attacks.

Case 11.—This 53-year-old man presented a six weeks' history of numbness in the left side of his face and tongue, followed later by clumsiness in the left fingers which progressed to a complete paralysis involving the entire left upper extremity. This paralysis cleared up to some extent, but numbness and weakness appeared in the left foot. The hyperalgesia disappeared in the face, but rather suddenly the entire lower extremity became completely paralysed. When admitted in the hospital he had a spastic left hemiparesis with complete paralysis of the lower extremity. There was an analgesia over the left extremities. Vibratory and position senses were absent in the left toes and fingers and there was a complete astereognosis in the left hand. The deep reflexes were hyperactive on the left side and he had a left Babinski sign. While in the hospital he developed convulsive movements which involved the left lower extremity. His blood-pressure was 138/76 and the blood serology negative. The pulse of the dorsalis pedis and the posterior tibial arteries was absent on the left side and the left foot was colder than the right. The left radial pulse was very weak and it was not possible to take a blood-pressure reading on the left arm.

Additional history revealed that for several years preceding his actual symptoms he noticed tiredness and pain in the calves when walking and he had to rest frequently. The capillaries in the nail-beds showed many abnormal forms and there was almost a complete stasis even after immersion of the hands in hot water. Plethysmographic studies showed greatly diminished pulsations in both the right and left fingers with only slight increase after immersion in hot water. Oscillometric tracings were greatly diminished in both legs and arms, but more so on the left side. Encephalography revealed dilated ventricles, the right being larger than the left, and symmetrically enlarged subarachnoid spaces. A diagnosis of cerebral and peripheral thrombo-angiitis obliterans was made. The patient died of a collapsed left lung following extirpation of the right stellate ganglion in an attempt to dilate his cerebral vessels.

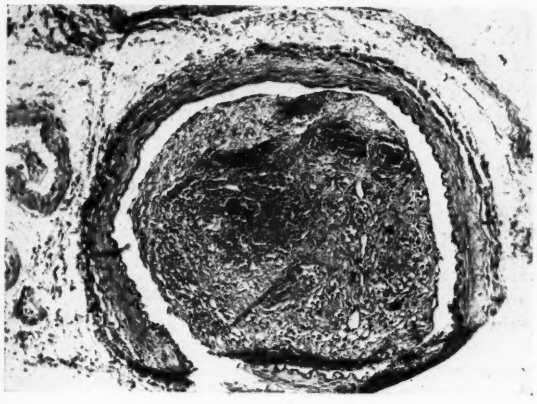
Autopsy revealed atrophy of both hemispheres with granular appearing gyri in the frontal, parietal, and temporal areas, especially on the right side. Numerous small, white, obliterated, distal arteries were seen in these areas and the arachnoid around these vessels appeared opaque and thickened. The major portions of the anterior, medial, and posterior cerebral arteries and their main branches were normal and delicate in appearance. The circle of Willis showed no abnormalities or evidence of sclerosis. The left internal carotid artery, however, was completely obliterated in its bony canal, but there was no gross evidence of atherosclerosis. Obliteration of the left axillary and iliac arteries was also found. Microscopically they showed an enormous endothelial proliferation in the subintimal layer, with complete obliteration of the intima and the lumen of the vessel. The media and the externa were intact and showed no calcium or other atheromatous deposits.

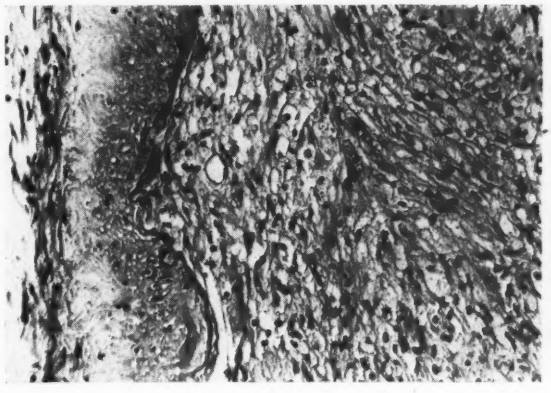
DISCUSSION

In 9 of the 11 cases with a similar clinical picture an osteoplastic craniotomy was performed. The dura mater was loose and baggy, the arachnoid thickened and opaque, and the subarachnoid space distended with cerebrospinal fluid. There were no signs of increased intracranial pressure. The distal portion of many of the smaller pial arteries in the exposed area of the brain was white and obliterated for a distance of one to two centimetres. Proximal to these portions of complete obliteration, the vessels were normal in size and appearance and contained liquid blood. These changes were observed in many of the distal branches of the middle cerebral artery scattered over the frontal, parietal, and temporal lobes. The appearance of the obliterated

vessels was that of worm-like white strings. Most of the vessels involved were arteries, but more rarely similar changes were found in veins. As Spatz (1935) and Lindenberg (1939) have shown, this process remains localized in one hemisphere or may be distributed symmetrically over both hemispheres in the border zone formed by the distal branches of the anterior, middle, and posterior cerebral arteries.

Disseminated cortical areas were found in various degrees and stages of yellowish discoloration, softening, and atrophy. There the gyri were small and granular and the sulci enlarged and deepened.





FIGS. 458, 459.—Microphotographs of cross- and longitudinal sections of an obliterated vessel removed at operation. They show proliferation of the endothelial cells of the intima and complete obliteration of the lumen of the vessel. The elastica and the media are normal.

Smaller areas of softening were also present in gyri which had not undergone atrophy. These areas of granular atrophy and necrosis lay in the field of distribution of the obliterated vessels. Adjacent areas of cortex contained an increased number of capillaries and small vessels which gave the appearance of an attempt to establish a collateral circulation.

Cross- and longitudinal sections of obliterated vessels removed at operation showed a marked proliferation of the endothelial cells of the intima and the subintimal layers, with little or no changes in the media and the adventitia (*Figs.* 458, 459). In some instances the media was narrower than normal. Large thrombi in various stages of organization completely obliterated the lumen of the vessels and were fixed to the vessel wall at the site of the roughest endothelial proliferation. They were well-organized and made up of loosely connected cells which contained in their meshes rests of pigment, newly proliferated small vessels, some histiocytes and a few small cells resembling lymphocytes. There were no fresh perivascular infiltrations in our sections as described by some authors in early stages of the disease. Spatz (1935) and others saw leucocytic and lymphocytic infiltrations in the thrombus, the adventitia of the vessels, and also in the subarachnoid space surrounding them in freshly diseased vessels. In the vessels which we removed at the time of operation no fresh inflammatory reactions were visible. However, in our cases studied at autopsy there were fresh inflammatory reactions in the intima of obliterated vessels and also around the wall of capillaries in areas of fresh cortical necrosis. The areas of granular atrophy showed the presence of numerous small foci of softening with areas of glial proliferation which acted like a contracting scar. Similar macroscopical and microscopical changes were described by Spatz (1935), Lindenberg (1939), Luers (1942), Antoni (1941), Scheinker (1937), and others in reports on cerebral thrombo-angiitis obliterans.

The anatomical diagnosis is made by the presence of the characteristic small, white, worm-like obliterated vessels, the granular atrophy of the cortex, and the presence of thrombi associated with proliferation of the endothelial cells of the intimal and subintimal layers of the vessels. It is easily differentiated from arteriosclerosis which usually attacks larger vessels and is characterized by local deposits of yellowish concrements in the vessel wall, usually the media, and which produce an enlargement of the vessel. Thrombo-angiitis obliterans involves, for the most part, the smaller arteries of the convexity, while arteriosclerosis has a predilection for the larger basal vessels and the proximal portions of the cerebral arteries. Hines and Barker (1940) have described an arteriosclerosis obliterans which they characterize by a hyalinization of the media with areas of fibrosis, calcification, and deposits of cholesterin and lipoids in the vessel wall, which thus occludes the lumen. Emboli from an endocarditis usually produce metastatic foci of encephalitis. Luetic endarteritis and periarteritis nodosa are also accompanied by marked inflammatory reactions which are more marked in the media and the adventitia of the vessel. The granular atrophy is secondary to numerous miliary areas of necrosis which result from the deficient circulation. Obliteration of one internal carotid artery is occasionally seen in connexion with cerebral thrombo-angiitis obliterans and has been described by Sorgo (1939), Antoni (1941), and Sunder-Plassmann (1941). It is considered to be the result of secondary thromboses similar to the obstructions found in the brachial or femoral artery in cases of advanced peripheral thrombo-angiitis obliterans.

The clinical diagnosis is more difficult. Most symptoms are transient in the beginning, come in attacks, and recede completely. They become permanent only in later stages of the disease. They

began in most of our cases with sensory disturbances, which were at first subjective and became later more objective. They usually involved only one side. The sensory symptoms were followed by weakness and a transitory paresis, which later became permanent. Except for one case which developed a paraplegia, the symptoms remained hemiplegic in type. Visual disturbances were present in 6 cases; extremity. Personality and memory disturbances were observed in 5 patients. Of the 11 patients 7 were males and 4 were females and their ages ranged from 34 to 63 years. The blood-pressure was somewhat increased in 3 patients and all had negative serologic reactions in the blood.

Thrombo-angiitis obliterans was diagnosed in 3 cases; in one the fundoscopic examination revealed

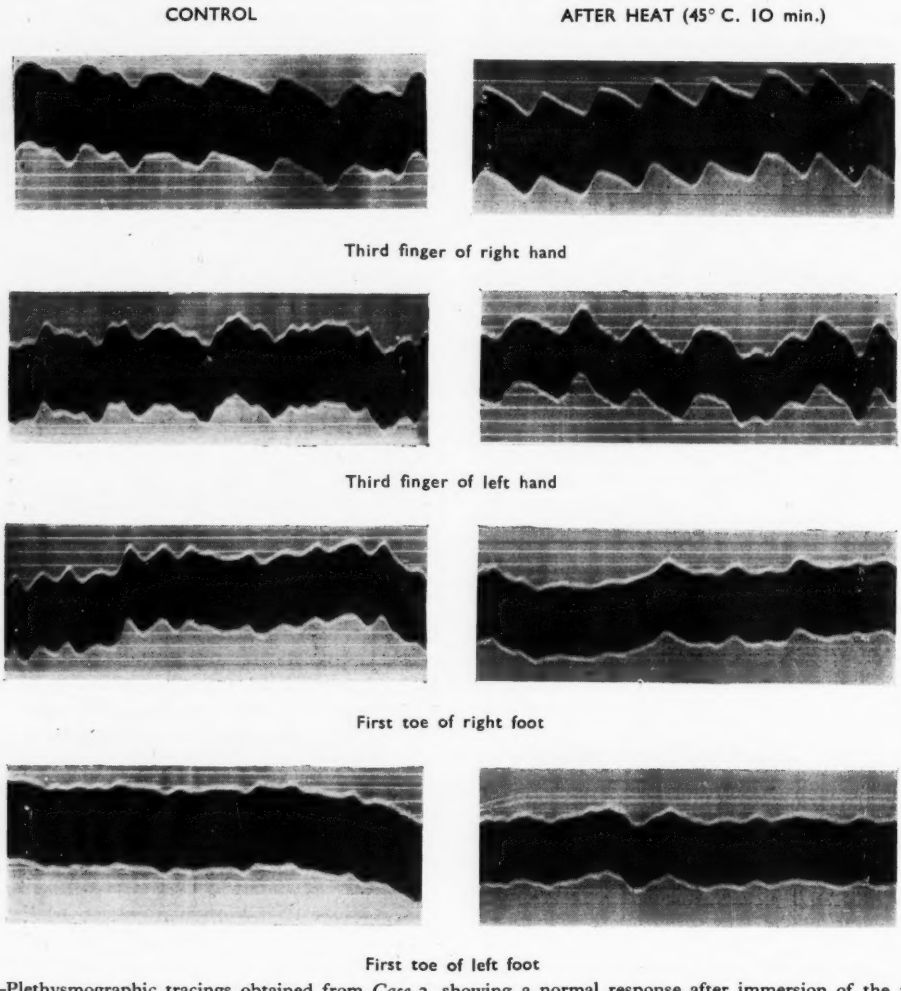

CONTROL
AFTER HEAT (45° C. 10 min.)
Third finger of right hand
Third finger of left hand
First toe of right foot
First toe of left foot

FIG. 460.—Plethysmographic tracings obtained from *Case* 3, showing a normal response after immersion of the patient's upper extremities in warm water for ten minutes, but no obvious increase in the amplitude of pulse volume in the lower extremities after immersion in warm water.

they were first noticed by the patient as transient disturbances and were occasionally associated with headaches of a migraine type. In 4 they resulted in a permanent homonymous hemianopsia. Speech disturbances were also common; they occurred in 6 patients, but remained incomplete and transient. In 3 of the patients the speech disturbance was associated with left-sided sensory and motor symptoms which pointed to the involvement of both cerebral hemispheres in the disease. This is made quite evident by the symptoms of the fourth patient, who had a severe paraplegia. Disturbance of stereognosis was present in 3 patients, 2 of whom otherwise had only slight sensory changes. Three patients had convulsive seizures which were generalized and associated with loss of consciousness and in 2 others the convulsions were localized in one

the presence of several arteries which were small, white, and obliterated in their distal portion. In another patient (*Case* 7) there were vascular changes in the skin; her extremities appeared mottled and were usually cold. The third patient (*Case* 11) had a history of intermittent claudication, and had obstructive vascular signs in two of his extremities. In the other 10 cases, however, the pulses of the radial, posterior tibial, and dorsalis pedis arteries were palpable and strong. None of the patients complained of disturbances in other organs, such as the heart, kidneys, and stomach, or of symptoms which could be interpreted as 'intermittent claudication of internal organs'. Air studies were performed in only 6 patients. In 3 the encephalograms showed definite evidence of cerebral atrophy with enlargement of the ventricular system and of the subarachnoid spaces

which ruled out a space-occupying lesion. In *Cases* 3, 4, and 10 the ventriculograms were not conclusive; the ventricles were slightly larger than normal and the cerebral atrophy was not outstanding. Sunder-Plassmann (1941) and Sorgo (1939) have pointed out the changes produced by cerebral thrombo-angiitis obliterans in arteriograms of the intracerebral vessels and think that this procedure is of some value especially in the diagnosis of advanced cases in which air studies are not conclusive. Arteriography was not performed in any of our cases.

Plethysmographic and oscillometric studies, which may show peripheral circulatory disturbances, were done in 3 cases. They failed to show any abnormalities in 2 cases in which there was no clinical evidence of peripheral Buerger's disease (*Fig.* 460). In *Case* 11 the changes were significant of a marked circulatory disturbance in all the extremities and helped in the diagnosis. The same was true of the microscopical study of the capillaries and the skin temperature changes following immersion of the hands in hot and ice water. However, the signs and symptoms of the other 10 patients pointed exclusively to a cerebral lesion and their slow progression suggested in most cases a space-occupying lesion.

The clinical diagnosis of cerebral thrombo-angiitis obliterans rests upon the long duration of transient but progressive symptoms, evidence of cerebral atrophy in pneumo-encephalograms, and possible peripheral vascular changes to be seen in the eye-grounds, in plethysmographic and oscillometric studies, or under the capillary microscope. It is true that in most cases peripheral vascular disturbances are not present and many of the latter tests will not be helpful in the diagnosis. The long duration of the symptoms and their transient character in the early stage of the disease suggest a circulatory disturbance; in these cases pneumo-encephalograms should always be done, and if they show the presence of dilated ventricles, unilateral or bilateral, without displacement, deformity, or defects, and evidence of enlarged subarachnoid spaces over the cerebral cortex, a space-occupying lesion may be ruled out and the diagnosis of a cerebral circulatory disorder can be made.

The aetiology and pathogenesis of cerebral thrombo-angiitis obliterans are the same as those of the peripheral forms. Abuse of nicotine, trauma, exposure to cold, and non-specific infections have been held responsible for the disease; but this could only be possible on the basis of a pathological vascular constitution. Roessle (1938) includes thrombo-angiitis obliterans with periarteritis nodosa in the group of the rheumatic vascular inflammations and thinks that the blood-vessel changes are part of a generalized allergic reaction. It is not definitely known whether the endothelial damage of the intima or the thrombus formation is the primary process, although it seems probable that a lesion of the intima exists prior to the formation of the thrombus. Most authors believe that the endothelial lesion causes a necrosis in the intimal and subintimal layers and results in the proliferation of endothelial cells upon which the thrombus forms. It is conceivable that the primary lesion may be the result of angiospasms since they have been observed in the retinal vessels by Marchesani and others. The anatomically proven changes cannot explain all the clinical disturbances. Many of the transient symptoms must be produced by temporary circulatory disturbances which cause only slight and reversible cerebral damage. Temporary decompensation of the collateral circulation and variations in the blood-pressure and in the amount of circulating blood may be held responsible for many of the inconstant symptoms.

Vasodilator, antispasmodic, and anticoagulant drugs have not been used successfully, but improvements have been described following repeated intravenous injections of saline solution. Foerster (1933) and Sunder-Plassmann (1941) found marked improvement in the general condition and cerebral symptoms of 6 patients following the resection of both cervical sympathetic chains, and recommend it as treatment for cerebral thrombo-angiitis obliterans. Sunder-Plassmann examined histologically the resected sympathetic ganglia and described marked degenerative changes of the ganglion cells. These may, however, be the result of the operative trauma and are far from conclusive evidence.

SUMMARY

Eleven cases of cerebral thrombo-angiitis obliterans are presented with symptoms and signs similar to those produced by a slowly growing intracranial tumour. Only 3 patients had evidence of peripheral circulatory disturbances. The history is usually of long duration and characterized by predominantly unilateral, early transient, but progressive symptoms which result in permanent damage. Nine cases were verified at operation and the microscopical examination of some of the obliterated vessels removed showed changes typical of this disease. Necrosis and granular atrophy of the brain result from the deficiency in circulation. The clinical diagnosis rests upon the inconstant character of the symptoms, the evidence of cerebral atrophy in pneumo-encephalograms, and peripheral vascular changes.

REFERENCES

ANTONI, N. (1941), *Acta med. scand.*, **108**, 502.
BARRON, M. E., and LINENTHAL, H. (1929), *Arch. Surg.*, **19**, 735.
BENEDEK, L. (1936), *Z. ges. Neurol. Psychiat.*, **156**, 646.
BIELSCHOWSKI, M. (1936), *Ibid.*, **155**, 313.
CSERNA, S. (1926), *Wien. Arch. inn. Med.*, **12**, 213.
FOERSTER, O., and GUTTMANN, L. (1933), *Arch. Psychiat. Nervenkr.*, **100**, 506.
GRESSER, E. B. (1932), *Amer. J. Ophthalmol.*, **15**, 235.
HAUSNER, E., and ALLEN, E. (1938), *Ann. intern. Med.*, **12**, 845.
HINES, E. A., and BARKER, N. W. (1940), *Amer. J. med. Sci.*, **200**, 717.
JAEGER, E. (1932), *Virchows Arch.*, **284**, 526.
LINDENBERG, R., and SPATZ, H. (1939), *Ibid.*, **305**, 531.
LUERS, T. (1942), *Arch. Psychiat. Nervenkr.*, **115**, 319.
MARCHESANI, O. (1934), *Klin. Wschr.*, **1**, 993.
PERLA, D. (1925), *Surg. Gynec. Obstet.*, **41**, 21.
ROESSLE, R. (1938), *Virchow's Arch.*, **288**, 780.
ROSENHAGEN, H. (1939), *Ibid.*, **305**, 558.
SORGO, W. (1939), *Zbl. Neurochir.*, **4**, 161.
SPATZ, H. (1935), *Dtsch. Z. Nervenheilk.*, **136**, 86.
STENDER, A. (1936), *Z. ges. Neurol. Psychiat.*, **156**, 76.
STRAUSSLER, E., FRIEDMANN, R., and SCHEINKER, J. (1937), *Ibid.*, **60**, 155.
SUNDER-PLASSMANN, P. (1941), *Dtsch. Z. Chir.*, **254**, 463.

SHORT NOTES OF RARE OR OBSCURE CASES

SUPERNUMERARY BURSA OF PECTINEUS MUSCLE SIMULATING HERNIA IN INGUINAL REGION*

By GORDON E. PERRIGARD†

DEPARTMENT OF ANATOMY, MCGILL UNIVERSITY, MONTREAL, CANADA

A HERNIA in the inguinal region may be simulated by many pathological conditions requiring differential diagnosis. One of these conditions which has not received much attention is the development of bursæ in this region. These bursæ generally occur in patients who have had a rather strenuous occupation which may have subjected this area to repeated trauma. The following case is a typical example.

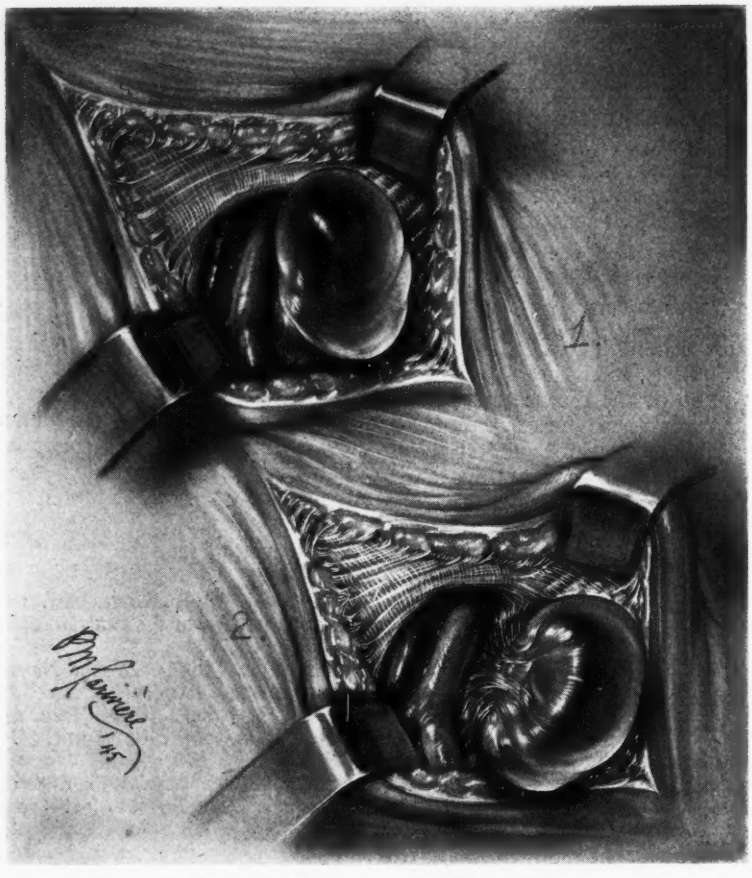


FIG. 461.—A supernumerary bursa of the pectineus muscle, medial to the femoral vessels, simulating a hernia in the inguino-femoral region.

CASE REPORT

HISTORY.—Mr. O. G., a referred case of 55 years of age, presented himself for "a hernia operation". He complained of a "rupture" in the right groin. He was a professional dancer and had practised acrobatic dancing for a number of years. There was no definite history of any injury to the groin.

ON EXAMINATION.—Regional examination revealed a mass about the size of a hen's egg subjacent to the medial half of the right inguinal ligament. It was round, firm, and slightly tender. There was no definite impulse on coughing, and it could not be reduced. It faintly transilluminated light. Examination revealed no connexion with the inguinal canal or the joint capsule. History revealed that it had gradually increased in size during the past two years. There was no regional lymphadenopathy. The patient had large external and internal hæmorrhoids. These were removed at the same time as operation on the groin.

AT OPERATION.—Since it was not thought to be a hernia, an incision was made inferior to the inguinal ligament over the mass. A large encapsulated cystic tumour was found which was firmly fixed to the fascia of the pectineus muscle subjacent to the inguinal ligament

* Paper presented at Ruddick Memorial Hospital, Montreal, March 13, 1946.

† Cooper Grant for Medical Research.

to the medial side of the femoral vein, and its capsule was adherent to several small branches of the long saphenous vein. Its gross appearance was that of a large ganglion of the type associated with tendon-sheaths (*Fig.* 461).

THE SPECIMEN.—

Gross: The specimen consisted of an oval cystic structure measuring 3·5 × 2·5 × 2·5 cm. It was thin-walled, the external surface being covered with a small amount of fibrous connective tissue and fatty tissue. A fatty and fibrous tissue was adherent to the point where the wall was thicker than elsewhere. It contained clear watery fluid. There was a small daughter cyst measuring 0·5 cm. in diameter.

Microscopical: Section of the cyst wall showed it to be composed of a thin layer of collagenous connective tissue lined with a single layer of cuboidal cells. The cytoplasm of the cells was pale pink, the nucleus a dark purple. The connective tissue showed two lymphoid follicles, one of which showed a germinal centre.

DISCUSSION

Especially in a ' referred ' case of possible hernia in the inguinal region, the pre-operative examination should include three things :—

1. Determine if a hernia actually does exist. Type it if possible.

2. Determine always if there is more than one hernia present.

3. Determine the nature of associated regional pathology (*a*) with, or (*b*) without a hernia.

Cases for Differential Diagnosis.—Out of the past 100 cases examined for hernia, several interesting conditions have been revealed. One woman had a cyst of the round ligament simulating a hernia. One man of 68 years of age had a direct inguinal hernia, but with a regional lymphadenopathy which turned out to be lymphosarcoma on pre-operative biopsy. Operation was deferred. This was three years ago, and he is still working. Lymphadenitis of the inguinal glands is very common in the labouring class, with the erroneous complaint of ' rupture '. Four cases of ' traumatic ' appendicitis have been operated on with the appendix in the hernia sac.

Frequently a lipoma precedes a hernia sac and such an accumulation of fat should always be removed or the capsule well incised to eliminate a possible contributory element in possible future recurrence of the hernia. During the war a combat pilot was referred for examination complaining of sudden severe abdominal cramps followed by vomiting, weakness, sweating, and pallor. Examination revealed acute tenderness in the left inguinal canal, with a small ¾-in. mass just distal to the internal inguinal ring. Immediate operation revealed an extraperitoneal lipoma which had been forced past the sharp edge of the deep inguinal ring, and had wedged a loop of small bowel in the inguinal canal in a small hernia sac.

The possibility of a coexisting hernia should be considered in any hernia repair. The development of a femoral hernia following an inguinal repair occasionally occurs. This may be due to the surgeon failing to detect the femoral hernia at the time of the inguinal hernia operation, or to a faulty technique in the hernia repair. The most common error is suturing the conjoined ' tendon ' to the inguinal ligament, and not to Cooper's ligament.

Bursæ may occur almost anywhere in the body in the region of muscles and tendons which are subjectable to trauma. The possibility of a supernumerary bursa of the pectineus muscle is another condition to add to the long list for the differential diagnosis of hernia in the inguino-femoral region.

CONGENITAL ATRESIA OF SMALL INTESTINE

BY F. DARNE

UNIVERSITY COLLEGE HOSPITAL

THE following case, being a rare type of atresia of the small intestine in the region of the middle of the midgut, may prove of interest.

CASE REPORT

HISTORY.—Full-term male infant, 2 days old, admitted to University College Hospital from Bromley Maternity Hospital with a history of vomiting after every attempt at feeding since birth. The vomit was of small bulk, yellowish in appearance, not projectile. The bowels had opened two or three times, a small amount of dark green material being passed.

ON EXAMINATION.—Full-term infant weighing 9 lb. 8 oz. T. 97°, P. 140, R. 40. Spina bifida occulta. Anterior fontanelle slightly depressed; slight amount of dehydration. Abdomen very distended, tympanitic. Very tense on palpation but not tender, no masses felt, no fluid. Rather weak and unnatural cry.

Rectal Examination: Little finger admitted without difficulty. No abnormality encountered. Rubber tube passed 6 in. without much difficulty; no passage of flatus or meconium.

X-ray Examination of Abdomen: The alimentary canal is distended by air; fluid levels are present in the stomach and in the small intestine. The colon and rectum are not outlined with air.

' *Meconium* ' film stained with methylene blue showed absence of the large vernix caseosa cells, thus pointing to complete atresia.

TREATMENT.—Dehydration combated with subcutaneous injections of saline.

Operation (F. D.): Day after admission (infant 3 days old). No premedication — local anæsthesia with 2 per cent novocain and adrenaline. Transverse incision through both recti and extending more laterally, immediately above the umbilicus. This gave beautiful exposure of the abdomen.

The gut was normally situated; no disorder of rotation. The stomach was distended and so was nearly the whole of the small intestine to a moderate degree. About 2 in. above the ileocæcal valve there was a complete interruption in the continuity of the small intestine and its mesentery, the gap corresponding to the middle of the midgut where Meckel's diverticulum is situated. The distal end of the proximal segment came suddenly to an end with a free border to its mesentery: for 8 in. it was enormously distended. The last 2 in. of the small intestine proximal to the ileocæcal valve was of the size of an adult ureter and there was a free border to its mesentery

at the upper end. The colon was of small diameter but appeared normal.

Procedure: The last 8 in. of the intestine above the gap were so distended that it had to be resected. The end was closed and invaginated: a side-to-side anastomosis was then performed between the small intestine immediately above the resected part and the ileum above the ileocæcal valve, using atraumatic 'arterial' silk in two layers, a through-and-through and a serous one. The abdomen was closed with catgut without drainage.

FIG. 462.—Radiograph showing air distending the alimentary canal. Fluid levels are present in the stomach and small intestine.

Post-operative: A small tube was passed through the nose into the stomach and aspirations carried out. Saline was continued by the subcutaneous route and this was eventually replaced by the intravenous way, using the long saphenous vein above the ankle.

PROGRESS.—The bowels worked two days after the operation and the abdomen remained soft. Hartmann's solution was then given by mouth. On the third and fourth days after the operation the infant seemed to be progressing satisfactorily, the bowels were working, showing that the anastomosis was functioning. On the fifth day after the operation the respiratory rate started to rise and the general condition deteriorated. He died on the sixth day.

FIG. 463.—Mesenteric aspect of distended small intestine immediately above atresia; note free edge of mesentery.

The immediate cause of death was bronchopneumonia, but no post-mortem examination was performed.

I wish to express my thanks to Mr. E. K. Martin, under whose care the infant was, for permission to publish these notes.

ACTINOMYCOSIS OF THE LIVER

REPORT OF AN UNUSUAL CASE

BY G. L. W. BONNEY

SURGICAL REGISTRAR, E.M.S. HOSPITAL, HAREFIELD

THE following account records an unusual instance of hepatic actinomycosis complicated by bilateral pleural and possibly pulmonary involvement. So far as may be gathered from the literature on this previously almost uniformly fatal disease, a case similar in pathogenesis, course, and response to specific treatment has not previously been reported.

CASE REPORT

A man of 39, a stockbroker before the war and during it an Army officer, had his appendix removed in 1925 at the age of 19 for acute appendicitis with peritonitis. Drainage of the peritoneal cavity was necessary and convalescence stormy, but he eventually made a good recovery and was well until December, 1943. At this time he had a right pleurisy with effusion and with purulent sputum, which cleared up in six weeks after treatment with sulphapyridine. There was recurrence of pleurisy and fever associated with multiple joint pains and polyarticular swellings in February, 1944, at which time a diagnosis of rheumatic fever was made. This illness was terminated after one month by the coughing up, over ten days, of 5 oz. daily of greenish foul-smelling sputum, and he was well until June, 1944, when there was recurrence of the joint swellings associated with

pleurisy, this time on the left side. With this, there was cough, purulent sputum, pyrexia up to 101°, loss of weight of 2 st., and occasional pain in either shoulder on deep breathing. Apart from the shoulder pain there were no symptoms referable to abdominal visceral disease and the story appeared to point to a localization of disease primarily in the chest.

The patient was admitted to this hospital under the care of Professor. G. W. Pickering on Sept. 9, 1944.

ON EXAMINATION.—A pale, thin, feverish man, pyrexia fluctuating up to 102°, pulse 90–100, respirations 30.

Respiratory system: Left pleural effusion up to 4th rib posteriorly, right chest clear.

Cardiovascular system: No evidence of cardiac failure or enlargement or of valvular disease.

Digestive system: Some carious teeth. No other throat lesion.

Abdomen: Fully healed Battle's incision of previous appendicectomy. Liver palpably enlarged 1 in. below costal margin in anterior axillary line, smooth, with some tenderness and spasm of overlying muscles. On one occasion a tender lump 2 × 1 in. could be detected on the anterior surface of the liver. No other abdominal lesion was demonstrated, diaphragmatic movement both sides was good, and rectal examination and examination of the urogenital system were negative.

Investigations :—

Pleural aspiration (repeated four times): Opalescent yellow fluid. culture sterile, cells lymphocytes and polymorphs, protein 3·9 mg. per 100 c.c. No T.B. on slide or culture. Sputum: No T.B. Culture: Pneumococci.

Urine: No abnormality.

Blood: Culture sterile on two occasions. Total plasma-proteins 6·2 g. per 100 c.c. W.B.C. 22,300 (polymorphs 78 per cent, lymphocytes 18 per cent, monocytes 4 per cent). (This polymorph leucocytosis persisted throughout.) Hæmoglobin 72 per cent. R.B.C. 3,850,000.

Mantoux test: Negative to 1–1000.

Radiographs: Left pleural effusion with no evidence of underlying lung disease. Right chest clear except for slight peaking of diaphragmatic pleura. Subsequent chest radiographs showed gradual diminution of the left pleural effusion without revealing underlying lung disease.

Barium meal and excretion pyelography showed no significant abnormalities.

TREATMENT AND PROGRESS.—Two 7-day courses each of 55 g. sulphadiazine were given on Sept. 22 and Oct. 14. Each produced the same effect—a delayed fall in the general level of the temperature and an improvement in the clinical condition. Both reverted about 10 days after stopping the drug to their previous state. Between Nov. 1 and Nov. 6 he was given 500,000 units of penicillin by continuous intramuscular drip—this had no effect upon the temperature, the clinical condition, or the leucocytosis.

Before, during, and after these therapeutic essays search was made in order definitely to localize the site of the hypothetical infective process. The presence of a swinging temperature with a persistent polymorph leucocytosis pointed to the presence of an infective lesion, but as the left pleural effusion receded it became increasingly clear that there was no definite evidence of lung disease. The recurrence after sulphadiazine treatment of right shoulder-tip pain with occasional hiccup, together with the persistent tender enlargement of the liver, was suggestive of infradiaphragmatic suppuration, but exhaustive clinical and radiological examination to confirm this yielded no clear evidence to support the diagnosis. It was eventually decided, in consultation with Sir Thomas Dunhill, that, in view of the continued fever and emaciation combined with the shoulder pain and liver enlargement, the evidence was enough to justify laparotomy in search of an as yet undiagnosed subphrenic lesion.

On Jan. 8, 1945, laparotomy under general anæsthesia was performed by Sir Thomas Dunhill.

The liver was exposed through a right upper paramedian incision and was seen to be free above from the diaphragm as far back as the attachment of the right coronary ligament, presenting on its upper surface about five small hard white nodules, with one fluctuant area 2 × 2 in. on the anterior part of the upper surface. The spleen and appendix area were surrounded with dense adhesions, but no other intra-abdominal lesion was found. One nodule from the liver was excised for section and the abdomen closed.

Aerobic culture of the nodule was sterile, while section showed the histology typical of actinomycosis, with the ray fungus clearly displayed.

Definitive treatment was started on Jan. 20, 1945, by:

1. Sulphamerazine 225 g. in 6-hourly 1-g. doses between Jan. 20 and March 19.

2. Penicillin 2,400,000 units by intramuscular drip between Feb. 18 and March 5.

3. Potassium iodide 15 gr. daily from Jan. 17 to March 3 and 45 gr. daily from March 6 to March 19.

The maximum blood-level of sulphamerazine reached was 7·5 mg. per 100 c.c. (free) on Feb. 9.

With this treatment the patient's general condition improved slowly, he became apyrexial on March 2 and a sinus forming at the upper end of the wound (from which *A. bovis* was grown on anaerobic culture) healed by March 10. On discharge, March 23, he felt quite well, had no chest or abdominal symptoms, his wound was soundly healed, and he had put on weight. Hæmoglobin was 80 per cent and W.B.C. 9800 (polymorphs 66 per cent).

Progress since discharge has been satisfactory: he gained 14 lb. in weight in the first month and when seen on Oct. 18 weighed 11 st. 6 lb. and felt quite well. The chest was clear on radiography and the liver appeared normal in size, though of a firmer consistency than is usual. Hæmoglobin 105 per cent, W.B.C. 8050. His latest report (March, 1946) is that he has no further symptoms, that his weight is maintained, and that he is able to lead a normal life. The abdominal wound remains soundly healed.

SUMMARY AND CONCLUSIONS

1. A case of hepatic actinomycosis presenting with a long history of recurrent attacks of pleurisy and causing great difficulty in diagnosis is reported.

2. The unusual pathogenesis and course is noted: liver involvement in this case may have been secondary to the appendicitis of twenty years ago or to lung disease. Bilateral pleurisy with effusion is noted as a complication not previously reported, as also is the small size of the lesion as seen at operation (possibly due to the previous treatment with sulphonamides).

3. The temporary beneficial effect of two inadequate courses of sulphadiazine and the lack of response to one insufficient course of penicillin is noted.

4. Recovery with maintenance of good health for over a year following treatment by massive doses of sulphamerazine and penicillin is reported.

My thanks are due to Professor Pickering and to Sir Thomas Dunhill for their permission to record this case, and for their continued interest in and help with this report. For histological, bacteriological, and other investigations I am much indebted to Dr. D. M. Pryce and Dr. C. N. Iland.

BIBLIOGRAPHY

(A selection from the literature available concerning the incidence and pathology of hepatic actinomycosis, and the response to treatment of actinomycosis in general, with particular reference to the effects of penicillin and the sulphonamides.)

ARIBAUD, G. (1897), *Lyon. méd.*, **85**, 491.
BANCROFT, F. W., and STANLEY-BROWN, M. (1905), *Ann. Surg.*, **41**, 641.
BARLING, B. (1944), *Lancet*, **1**, 503.
BENBOW, E. P. (1944), *J. Amer. med. Ass.*, **125**, 993.
BENNETT, J. G. (1944), *Brit. med. J.*, **1**, 197.
BEVAN, A. D. (1905), *Ann. Surg.*, **41**, 641.
BILLINGTON, R. W. (1944), *Brit. med. J.*, **1**, 326.
BUTLER, E. C. B. (1945), *Proc. R. Soc. Med.*, **38**, 481.
CHITTY, H. (1929), *Brit. med. J.*, **1**, 347.
— — (1944), *Ibid.*, **1**, 115.
CHRISTOPHER, F. (1940), *Amer. J. Surg.*, **50**, 371.
COPE, V. Z. (1938), *Actinomycosis*. Oxford Medical Publications.
CROHN, B. B. (1943), *Gastroenterology*, **1**, 140.
CUTTING, W. C., and GEBHARDT, L. P. (1941), *Science*, **94**, 568.
DEAN, C. W. A. (1917), *Brit. med. J.*, **1**, 82.
DIXON, G. J. (1939), *Ibid.*, **2**, 686.
DOBSON, L., and CUTTING, W. (1941), *J. Amer. med. Ass.*, **116**, 272.
DORLING, G. C., and ECKHOFF, N. L. (1940), *Lancet*, **2**, 708.
DUCKWORTH, D. (1900), *Brit. med. J.*, **2**, 1189.
GARROD, L. P. (1944), *Ibid.*, **1**, 328.
GOOD, L. P. (1931), *Arch. Surg.*, **22**, 307.
GREENWELL, A. W. (1928), *Mil. Surg.*, **62**, 185.
HALL, W. H. (1939), *J. Amer. med. Ass.*, **112**, 2190.
— — and SPINK, W. W. (1943), *Ibid.*, **123**, 125.
HAMILTON, A. J. C., and KIRKPATRICK, H. J. R. (1945), *Brit. med. J.*, **2**, 728.
HENDRICKSON, E. G., and LEHMAN, E. P. (1945), *J. Amer. med. Ass.*, **128**, 438.
HERRELL, W. E. (1944), *Ibid.*, **125**, 1009.
HOLLENBECK, W. F., and TURNOFF, D. (1943), *Ibid.*, **123**, 1115.
JACOB, F. H. (1944), *Brit. med. J.*, **1**, 266.
KEENEY, E. L., AJELLO, L., and LANKFORD, E. (1944), *Johns Hopk. Hosp. Bull.*, **75**, 393, 410.
Leading articles (1944), *Lancet*, **1**, 764; (1945), *Brit. med. J.*, **2**, 326.
LYONS, C. (1943), *Surgery*, **14**, 99.
— — (1943, a), *J. Amer. med. Ass.*, **123**, 312.
— — (1943, b), *Ibid.*, **123**, 1010.
MACGREGOR, A. B. (1945), *Proc. R. Soc. Med.*, **38**, 639.
MACNAB, G. H. (1945), *Edinb. med. J.*, **52**, No. 6, 219.
MILLER, E. M., and FELL, E. H. (1939), *J. Amer. med. Ass.*, **112**, 731.
MORTON, H. G. (1940), *Canad. med. Ass. J.*, **42**, 231.
MYERS, H. B. (1937), *J. Amer. med. Ass.*, **108**, 1874.
OGILVIE, W. H. (1940), *Brit. med. J.*, **2**, 254.
POULTON, E. P. (1937), *Proc. R. Soc. Med.*, **31**, 164.
ROBERTS, J. E. H., and TUBBS, O. S. (1945), *Lancet*, **1**, 39.
SHEARBURN, E. W. (1943), *Surgery*, **13**, 38.
WALKER, O. (1938), *Lancet*, **1**, 1219.

CALCIFICATIONS IN LIVING TISSUES

BY JOHN T. MORRISON, LIVERPOOL

FOUR examples are recorded of calcification of tissues, of clinical interest in themselves and as illustrations of this curious pathological process.

The first is taken from an old woman in whom hard nodules were discovered in both breasts in the course of a routine examination. The stony

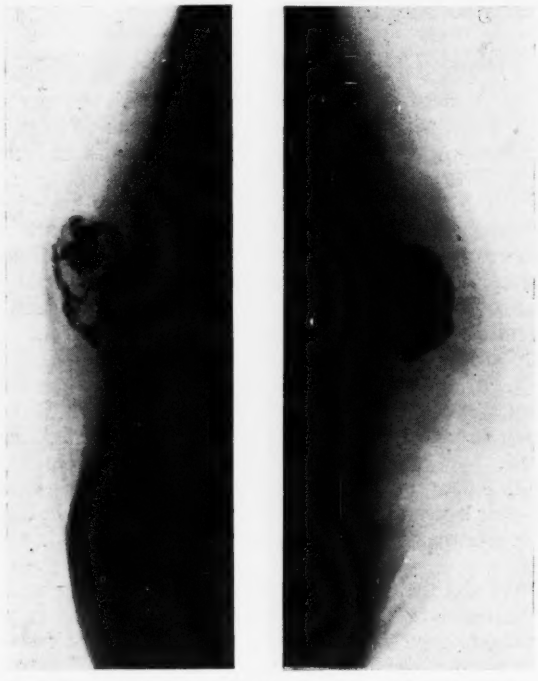


FIG. 464.—*Case* 1. Lateral view of calcified fibro-adenomata in both breasts.

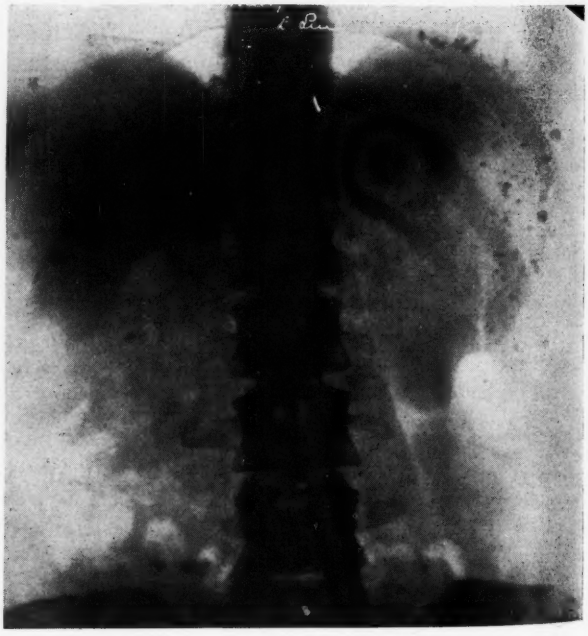


FIG. 465.—*Case* 2.—Numerous calcified masses in the liver and spleen The outline of the lower pole of the latter is clearly seen, and the crescentic distribution of the masses in the spleen is characteristic.

hardness at once aroused suspicion as to the diagnosis and this was confirmed by lateral X-ray views of the breasts (*Fig.* 464).

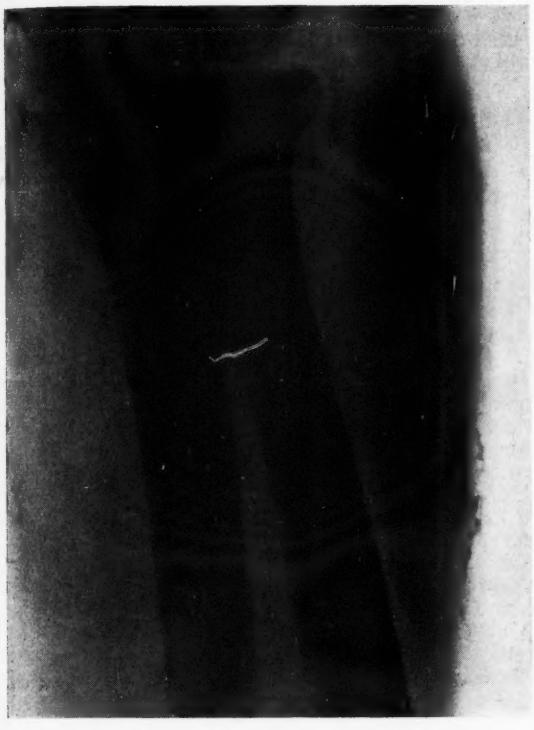


FIG. 466.—*Case* 3. A tangential view of the forearm showing the calcification in the superficial scars.

The second is the radiograph of an abdomen taken in the course of investigation of a woman of 48 suffering from urinary tuberculosis (*Fig.* 465). Right nephrectomy had been performed some years earlier. Symptoms had now recurred as the result of infection of the left kidney. There was nothing in the clinical history to suggest at what date the extensive tuberculous disease in the liver and spleen had occurred.

FIG. 468.—*Case* 4. Lateral view, showing the very superficial position of the calcified area.

The third is a photograph of a young man who, in early childhood, had suffered from diphtheria. After injections in the forearms suppuration had apparently followed—as witness the incisions—and now the scars are broad, smooth, pinkish, depressed, and bony-hard to the touch (*Figs.* 466, 467). The condition gives rise to no disability, and so far no trophic disturbances over the calcareous deposits

FIG. 467.—Forearms of *Case* 3, showing scars in which calcification had taken place.

have been noted. The Wassermann reaction was strongly positive, but this is presumably merely a coincidence.

The fourth is a lateral radiograph of the foot of a child of 6, a patient of Dr. W. A. Mackay, of Southport, to whom I am indebted for the photograph. The calcified nodule was covered by thin adherent skin (*Fig.* 468). The mass was excised for histological examination. It proved to be a benign calcifying epithelioma, with advanced degeneration and calcification so that only the ghosts of the epithelial masses remained. Another example of this peculiar kind of tumour was recently observed in the scalp of a child only eight months old; the general histological appearances were similar, but the epithelial masses were much less degenerate.

These are all, of course, examples of calcification in the classical conditions of diminished blood-supply. The subject is comprehensively discussed in the illuminating article by Watson-Jones and Roberts in *The British Journal of Surgery*, 1934, **221**, No. 83.

AN UNUSUAL CASE OF INTUSSUSCEPTION

By CLIFFORD JONES, M.B.E.
FIRST ASSISTANT, SHEFFIELD ROYAL INFIRMARY

Owing to the unusual causation of this case of intussusception, it has been considered worthy of being placed on record.

CASE REPORT

On Feb. 8. 1946, a male aged 54 was admitted to the wards of Professor R. St. Leger Brockman at the Royal Infirmary, Sheffield, complaining of acute colicky pain in the abdomen which was chiefly sub-umbilical in situation. There was a history of occasional pain in the right iliac fossa for five months previously, and this had been accompanied by some loss in weight and a feeling of general ill health. On the night of Feb. 7 he was seized with acute abdominal pain, colicky in nature, and had vomited twice. The bowels were opened on Feb. 6 but not since. There were no other relevant points in the history.

Fig. 469.—Photograph of the portion of intestines removed at operation, showing the ileocolic intussusception.

On Examination.—The patient was a thin spare individual, the abdomen was slightly distended, particularly in the the lower half. There was no visible peristalsis and nothing abnormal was heard on auscultation.

On palpation a hard mass was felt in the right iliac fossa, which was slightly tender. There was no rigidity.

On rectal examination nothing abnormal was found.

A provisional diagnosis of carcinoma of the cæcum was made.

During the next few days the bowels opened naturally and there was no pain. On Feb. 15 the patient had another attack of intense colicky pain. The abdomen was now very tender and difficult to examine, but was not grossly distended. There had been no vomiting. A tentative diagnosis of acute on chronic obstruction was made and a laparotomy was performed.

Operation (C. J.).—General anæsthetic: open ether. Right lower paramedian incision. On incising the peritoneum there was a copious flow of brownish odourless fluid in which there were many flakes of fibrin. On exploring the right iliac fossa a mobile mass was found which extended along the ascending colon and a proximal part of the transverse colon. The cæcum formed a cuff around the entering small bowel which could be readily entered with exploring fingers and it was then realized that it was an intussusception of the ileocolic type. Reduction was impossible and as the patient's condition was good, a right hemicolectomy was performed with an end-to-side anastomosis of ileum into transverse colon.

Convalescence was uneventful and the patient was discharged home on March 4.

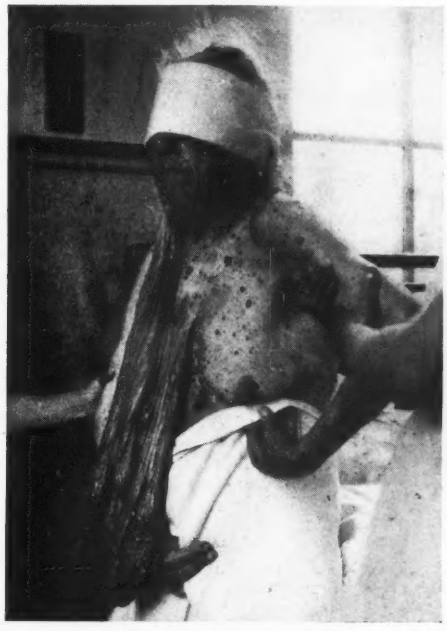
The Specimen.—Consists of the lower ileum, cæcum, and colon. The ileum is intussuscepted into the colon for 9·5 cm. At the apex of the intussusception there is a hard hæmorrhagic mass 10 cm. in length. This mass is arising from the wall of the ileum. The ileocæcal valve is grossly dilated and the mass had obviously passed through the valve, drawing the ileum after it. The entering and returning layers of the intussusception are covered with a fibrinous exudate and are adherent to one another. A large section of the mass shows a necrotic and hæmorrhagic surface, but with the structure of a fibroma. Scattered throughout the section are groups of plain muscle-fibres. It is, therefore, classed as a fibro-leiomyoma of ileum.

My thanks are due to Professor R. St. Leger Brockman for permission to publish the case and to Dr. L. C. D. Hermitte, Pathologist to the Royal Infirmary, for help in examining the specimen and for the photography.

FIBROMA PENDULUM

By N. DUGGAN, Worcester

In a recent number of this Journal (1944) I reported the case of a very large lipoma growing from the right side of the neck. The accompanying illustrations depict an even more fantastic incubus, complicating a case of neurofibromatosis and arising from the same area of the body. Sequeira (1927) gives the name 'fibroma pendulum' to these large pendulous tumours.



Figs. 470, 471.—Photographs showing the extent of the tumour.

Fig. 472.—Photograph showing the neurofibromatosis.

CASE REPORT

The patient, a healthy and intelligent woman of 72, stated that the condition was congenital. No other members of her family have been affected.

She recalls that in childhood the tumour was 6–8 in. long. It reached to her waist for most of her adult life, and has grown rather rapidly during the past two years. No great pain was felt, and the life-long burden was accepted as a matter of course. She has never consulted a doctor for any reason, until two years ago, when she fractured her clavicle, and the attending doctor suggested that operation might be worth consideration.

She was admitted to hospital in March, 1946, and several days were occupied in thorough pre-operative cleansing of the skin by cetavlon and sulphonamide cream.

At operation, which was performed entirely by diathermy, the pedicle, consisting mostly of hypertrophied platysma and dilated veins, was easily divided and the skin sutured to make a linear scar 9 in. long.

The wound, which was dusted before closure with penicillin and sulphonamide powder, healed well by first intention, except for a small central area where a rubber drain had been inserted. The tumour on removal weighed 7½ lb.

REFERENCES

Duggan, N. (1944), *Brit. J. Surg.*, **32**, 324.
Sequeira, J. H. (1927), *Diseases of the Skin*, 51.

CASE OF SARCOMA OF STOMACH SIMULATING SPLENIC TUMOUR

By B. WOLMAN

RESIDENT MEDICAL OFFICER, WITHINGTON HOSPITAL, MANCHESTER

GASTRIC sarcoma is a sufficiently rare lesion to warrant the recording of any new case. The following case, seen at Ancoats Hospital, provided an interesting problem in diagnosis.

CASE REPORT

The patient, a married woman aged 34 years, was admitted to hospital on Jan. 20, 1943, complaining of a large, painful swelling in the left side of the abdomen of nine to ten weeks' duration. The pain, situated over the swelling, was continuously dull and aching, but gave rise to acute exacerbations of griping nature. Appetite

FIG. 473.—Showing external appearance of stomach, colon, and mesentery.

was good. There was no nausea or vomiting. Her bowels, always constipated, had not been increasingly so during the present illness. There were no urinary complaints, but her menses had stopped seven months previously. She was losing weight, but had no idea of the exact amount.

Her previous history was unimportant apart from the fact that she had " rheumatism " when she was a child.

ON EXAMINATION.—The patient was frail, thin, and obviously very poorly. The mucosæ were very pale, the tongue was clean, and the throat healthy. There were no glands palpable in the neck, groins, or axillæ.

The heart was enlarged to the left, with a definite auricular fibrillation—about 100 per minute. Blood-pressure was 120/70 and there were no abnormal clinical signs in the lungs.

The abdomen was distended and showed the patchy pigmentation of erythema *ab igne* over the whole of the left side. There was a large tumour extending from below the left costal margin towards the midline and reaching down as far as the level of the iliac crest. It was impossible to get between the mass and the costal margin, but it was obvious that the tumour was not coming out of the pelvis. The tumour was superficial and tender. It had a smooth surface and irregular anterior edge, although there was no definite notch. The liver edge, distinct from that of the tumour, was palpable and found to be regular and smooth. There was no œdema and no ascites.

DIAGNOSIS.—Clinically, the mass resembled an enlarged spleen. True, it was tender, and had an irregular anterior border and no definite notch, but a possible diagnosis was perisplenitis and infarcted spleen due to recurrent emboli resulting from auricular fibrillation.

The length of the history and the fact that the condition was not settling were against the acceptance of this diagnosis.

The coexistence of anæmia, enlarged liver, and enlarged spleen suggested pernicious anæmia as a diagnosis. This was confirmed by a peripheral blood-count of the macrocytic hyperchromic type as follows :—

Hæmoglobin 50 per cent.

Red blood-corpuscles 1,820,000 per c.mm.

Colour index 1·38.

White blood-corpuscles 5000 per c.mm.—in normal proportions.

There was 1 per cent of nucleated red blood-cells, and marked anisocytosis and poikilocytosis.

Examination of the sternal marrow was as follows :—

	per cent
Hæmatoblasts	2·5
Megaloblasts	7·75
Normoblasts	8·5
R.B.C. nucleated	6·0
Lymphocytes	8·5
Monocytes	0·25
Myeloblasts	3·5
Myelocytes	10·25
Metamyelocytes	18·5

Although a fractional test-meal revealed complete achlorhydria, the extreme tenderness of the 'splenic' swelling contra-indicated the diagnosis of pernicious anæmia.

The possibility of a gastric or colonic neoplasm was considered, but unfortunately the patient was far too ill to stand a barium meal and gastric X-ray examination. A renal lesion was eliminated by a normal intravenous pyelogram.

It was decided to give a short test course of daily liver injections (Lederle Liver Preparation 3 c.c.), together with iron and hydrochloric acid, but after two weeks there had been no reticulocyte response and, in fact, the hæmoglobin had fallen to 44 per cent.

LAPAROTOMY.—In view of the vagueness of the diagnosis and the demand by the patient that something drastic should be done to relieve her suffering, an exploratory laparotomy was performed after an emergency blood transfusion had been given.

The mass was not splenic but gastric in origin and consisted of a neoplasm of the lower half of the body of the stomach, around which was wrapped the greater omentum containing numerous secondary deposits. There were metastases throughout the peritoneum, but the liver, although enlarged, was free of secondary

growth. The abdomen was closed. The patient collapsed suddenly on the third day after operation and died.

AT AUTOPSY.—The above findings were corroborated. The liver was enlarged and showed passive venous congestion. The spleen was normal in size, but there

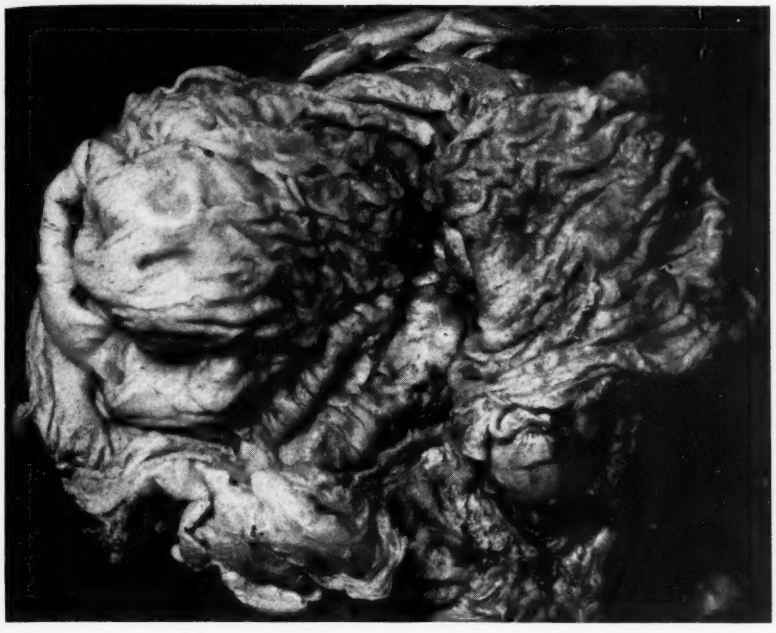


FIG. 474.—Stomach opened to show normal mucous membrane.

was a tumour mass extending into the hilum. The tumour itself involved the wall of the lower half of the body of the stomach, and the omentum and mesentery, studded with many hard deposits, were wrapped around it so as to simulate a tumour much harder and larger than actually existed. There were dense adhesions between the mass, the anterior abdominal wall, and all surrounding viscera. The cavity of the stomach and the mucosa appeared to be normal (*Fig.* 474). Microscopical examination of the tumour revealed a spindle-celled sarcoma (*Fig.* 475). The remaining viscera were not examined, as permission was only given for abdominal autopsy.

DISCUSSION

Early case records of sarcoma of the stomach were reported by Warner (1917) and Barrington-Ward (1916); and Haggard reviewed 244 cases in the literature in 1920. It occurs infrequently, Ewing (1940) estimating that sarcoma appeared as only 1 per cent of gastric tumours. The Mayo Clinic reported a yearly incidence of 1 to 6 cases (Hurt and Broders, 1932).

The average age of incidence of the reported cases was 40 years, while that of carcinoma was 55 years. The sex incidence is about equal. The growth is commonest on the lesser curvature and is usually diagnosed as carcinoma. Sarcomata may be diffusely infiltrating or nodular in type. The spindle-celled variety is commonest. It is rarely possible to make a correct pre-operative diagnosis, although the X-ray picture may be suggestive. In an early case the treatment is resection of the growth, although the lymphosarcoma variety are reported to be sensitive to deep X rays.

The case reported here is interesting as a diagnostic problem: (1) on account of the character of the tumour itself; and (2) because of the other clinical findings. The development of a macrocytic hyperchromic anæmia of a pernicious anæmia type is interesting as it is not usually a feature of gastric sarcoma, although, of course, it is very common in carcinoma. Achlorhydria also is a variable sign.

Extreme tenderness of the tumour, coupled with the absence of nausea and vomiting, served as efficient pointers—away from the gastro-intestinal tract.

SUMMARY

1. A new case of gastric sarcoma is reported, together with autopsy and histological findings.

2. The interesting features in the differential diagnosis are discussed.

My thanks are due to Dr. G. J. Langley for permission to report the case, and to Dr. F. Duran-Jorda for the microphotographs.

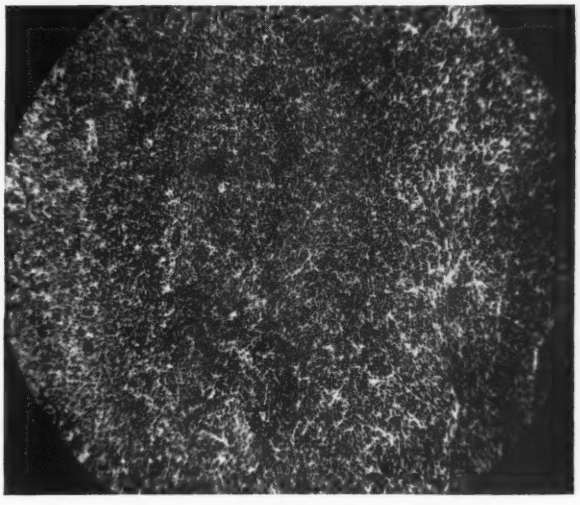


FIG. 475.—Microscopical appearance, showing spindle-celled sarcoma.

REFERENCES

BARRINGTON-WARD, E. L. (1916), *Brit. J. Surg.*, **4**, 301.
EWING, J. (1940), *Neoplastic Diseases*, 4th ed., 284. London.
HAGGARD, W. D. (1920), *Surg. Gynec. Obstet.*, **31**, 505.
HURT, H. H., and BRODERS, A. C. (1932), *Proc. Mayo Clin.*, **7**, 576.
WARNER, F. (1917), *Ohio med. J.*, **13**, 647.

A CASE OF CARCINOMA OF THE ILEOCÆCAL VALVE, WITH FORMATION OF A SPONTANEOUS ILEOCÆCAL FISTULA

By F. R. R. MARTIN

RESIDENT SURGICAL OFFICER, LEEDS GENERAL INFIRMARY

SPONTANEOUS formation of fistulæ is not uncommon in carcinoma of the large intestine. Lockhart-Mummery (1934) describes two ways in which they may arise: (1) The growth becomes adherent to an adjacent viscus and, as ulceration occurs, the central portion gives way, leaving a communication between the two. (2) The growth in the colon becomes ulcerated, and the ulcer perforates the bowel wall, forming a pericolic abscess, which, in its turn, ruptures into an adjacent viscus.

FIG. 476.—Photograph of the terminal ileum and cæcum, showing the carcinoma of the ileocæcal valve and the fistula.

The case described below is of interest because the fistula found in the immediate neighbourhood of a carcinoma of the ileocæcal valve cannot be said to have been caused by either of the processes described above. Furthermore, on looking through the literature, I can find no mention of a similar case.

CASE REPORT

T. T., aged 58, was admitted to the General Infirmary, Leeds, on Dec. 18, 1945, with symptoms of subacute intestinal obstruction. At laparotomy a small growth was palpated in the ileocæcal valve, the lumen of the gut being obstructed at this level. A considerable length of terminal ileum was dilated and hypertrophied. An anastomosis was made between the ileum and transverse colon. Unfortunately a low-grade peritonitis occurred and the patient died some five weeks later.

At autopsy the condition depicted in the accompanying photograph (*Fig.* 476) was found. The terminal ileum (right) and the cæcum and ascending colon (left) have been laid open, disclosing a nodular growth which completely encircles the ileocæcal valve, causing very great narrowing of the lumen. Further examination revealed the presence of a narrow fistulous track through the base of the ileocæcal valve, external to the growth, and apparently not directly related to it. The fistula was about 1·5 cm. in length and some 5 or 6 mm. in diameter. It was situated posteriorly to the natural lumen. A tapered glass rod has been passed through the fistula from the cæcal aspect.

Histologically, the tumour is a fairly well-differentiated adenocarcinoma, probably of comparatively slow growth. It has extensively infiltrated the muscular coat. Much inflammatory change is present in places in the stroma.

Comment.—The fistula had probably arisen by a simple ulcerative process resulting from the chronic obstruction.

Another instructive feature of this case is the fact that, in spite of the presence of the fistula, acute intestinal obstruction had developed. This finding is in agreement with the opinion of Lockhart-Mummery (1934), who states that the occurrence of fistulæ will not, as a rule, prevent the onset of obstructive symptoms.

My thanks are due to Professor Stewart for his advice in preparing this short note and to Mr. Digby Chamberlain for his permission to publish this case.

REFERENCE

LOCKHART-MUMMERY, G. P. (1934), *Diseases of the Rectum and Colon*, 538. London: Baillière, Tindall & Cox.

TUBERCULOSIS OF MECKEL'S DIVERTICULUM

By IAN B. MACDONALD

DEPARTMENT OF PATHOLOGY, UNIVERSITY OF TORONTO

TUBERCULOSIS of Meckel's diverticulum is exceedingly rare, only 9 authentic cases having been reported in the literature. The earliest of these was recorded by Concato in 1871, and the most recent in 1932 by Paul Michael. Of these cases none was diagnosed clinically.

In 1809 Meckel described this congenital diverticulum as a remnant of the omphalomesenteric duct. Since that time an ever increasing list of pathological complications have been recognized, varying from heterotopic gastric or pancreatic tissue to ulcers, neoplasms, and mechanical obstructions. In recent

years surgeons have become increasingly aware of pathological changes in this structure as a source of abdominal crises.

The incidence of Meckel's diverticulum is 1 to 2 per cent, of which about 33 per cent display various abnormalities (Balfour, 1911). It is obvious, therefore, that in only a very small percentage of persons will this vestigial pouch be associated with pulmonary tuberculosis; about half of these will have intestinal tuberculosis, and tuberculous infection of the diverticulum will be most uncommon. Various complications may be superimposed upon this lesion, such as intestinal obstruction or perforation of the ulcer with a subsequent tuberculous peritonitis, and clinical evidence of these conditions may be produced.

PATHOGENESIS

Ulcerative tuberculous enteritis is generally regarded as a primary lesion in children, whereas in adults it is secondary to pulmonary tuberculosis. Primary tuberculosis in a Meckel's diverticulum has never been reported, and is apparently unknown. Tuberculous infection occurs rather as a lesion secondary to tuberculosis elsewhere in the body. The tubercle bacilli may either be carried to the intestines by the blood-stream from a focus in the lungs or swallowed in the sputum, and penetrate the intestinal mucosa to form a secondary lesion in the bowel wall. The latter process is the probable explanation of tuberculous diverticulitis, and is also applicable to infection of the appendix. Coley (1925) reports a case of simultaneous tuberculous involvement of the appendix and the diverticulum of Meckel.

CASE REPORT

Clinical History.—W. T., a white Canadian aged 65, was admitted to the Toronto General Hospital on Sept. 4, 1945. Nine months previously he had developed a cough productive of thick yellowish sputum. At the same time he began to manifest symptoms of cardiac failure, which increased in severity until the time of death. At no time was there any abdominal distress.

At Autopsy.—The heart was greatly enlarged, the foramen ovale was widely patent, and there was an aneurysm of the pulmonary artery. The upper lobes of both lungs, including the apices, and the right lower lobe contained numerous small caseous areas. No gross cavities were present. The bronchial and peribronchial lymph-nodes were normal.

A Meckel's diverticulum was present, measuring 3 cm. in length and arising from the ileum 55 cm. proximal to the ileocæcal junction. Its tip was attached to the parietal peritoneum by a few friable adhesions. The serosal surface of this diverticulum was dark red, with many white, pin-point nodules scattered diffusely over it (*Fig.* 477). Its lumen was patent throughout and continuous with that of the ileum. The mucosa was also dark red and an ulcer about the size of a 25-cent piece was present, the edges of which were rolled and had a velvety appearance. The base of the crater was firm, smooth, and of a similar colour. No other lesions of the gastro-intestinal tract were present.

Microscopically the pulmonary lesions showed a picture of active tuberculosis, consisting of patches of caseation surrounded by epithelioid cells, giant cells, and lymphocytes. The ulcer in the diverticulum was also tuberculous. The mucosa was necrotic and infiltrated with inflammatory cells. A few scattered tubercles were present in the subserosa.

Fig. 477.—Showing the dark red serosal surface of the diverticulum, with many white pin-point nodules scattered diffusely over it.

This patient died as the result of cardiac insufficiency due to congenital heart disease, with pulmonary tuberculosis as a contributing factor. This latter was regarded as the source of the tuberculous infection of the diverticulum.

SUMMARY

1. A case of tuberculous ulcer of Meckel's diverticulum is reported, secondary to pulmonary tuberculosis.

2. The ulcer was unaccompanied by clinical symptoms and there were no other tuberculous lesions of the gastro-intestinal tract.

3. This is the tenth case recorded since 1871.

REFERENCES

Balfour, Donald (1911), *J. Minnesota Med.*, **21**, 110.
Coley, B. L. (1925), *Arch. Surg.*, **11**, 519.
Michael, Paul (1932), *Ibid.*, **25**, 1152.

MULTIPLE CAPILLARY HÆMANGIOMA OF THE SKULL BONES

By A. S. HANDOUSA BEY

PROFESSOR OF OTORHINOLARYNGOLOGY, FOAD I UNIVERSITY, CAIRO, EGYPT

HÆMANGIOMA of bone is a very rare finding; the majority of the recorded cases are of the cavernous type. Mary S. Sherman (1944) reported a case of capillary hæmangioma which was situated in the distal metaphysis of the left femur, and could only find 4 other cases of the same type recorded in the literature.

FIG. 478.—Photograph of patient, showing the proptosis

In view of this extreme rarity of capillary hæmangiomata of bone I feel the following case is worth recording. It was discovered in the skull bones in two situations and was found to be the sole cause of the proptosis for which the patient sought medical advice.

CASE REPORT

Moh. A. T., male, aged 36 years (*Fig.* 478), complained of right-sided, gradually increasing proptosis of about one year's duration, associated with excessive lacrimation. There was no pain and no history of trauma or syphilis. The patient stated that in childhood he had a severe eye illness (? purulent ophthalmia), followed by loss of vision of his left eye.

ON EXAMINATION.—Clinically the right eye was markedly proptosed, mainly forwards and downwards, with increased vascularity of the upper lid and congestion of the conjunctiva, with slight limitation of the globe movements mainly upwards and outwards. There was a minute nebula on the right cornea (at 5 o'clock); the pupil reacted normally; intra-ocular tension was normal.

The superior orbital margin was markedly thickened, especially at its outer part, but not tender. An intra-orbital firm mass was felt in the outer orbital corner (? dislocated right lacrimal gland), which was slightly tender on pressure.

There was no pulsation felt over the frontal bone or the orbital contents; the proptosed structures were not reducible.

Right optic disk was swollen, with a hazy margin.

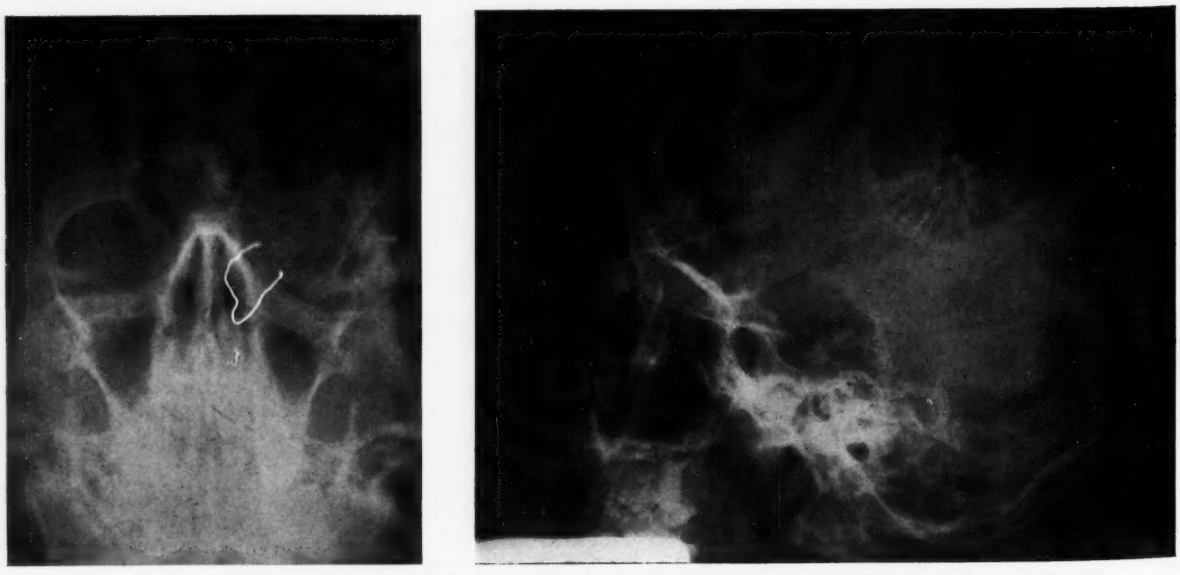
Right vision 6/36. Right field of vision showed concentric contraction in all directions.

Left eye showed diffuse keratitis, otherwise there was nothing special.

The skull showed no swellings or tender points.

There was right-sided maxillary and ethmoid sinusitis with nasopharyngitis.

Radiological Examination (*Figs.* 479, 480) showed two wheel-like shadows lying in and distending the



FIGS. 479, 480.—Radiographs showing shadows lying in and distending the diploic part of the bone between the two tables with bony trabeculæ radiating towards the periphery.

diploic part of the bone between the two tables, and was characterized by having bony trabeculæ radiating from a common centre towards the periphery. One of these shadows lay in the right side of the frontal bone and the other in the right parietal bone.

The X-ray investigation was first made by Dr. Sinna, to whom I am indebted for the preliminary diagnosis of hæmangioma of bone. Further X-ray investigations were kindly carried out by Dr. Mary Bey and Dr. Nissim Bey Abou-Saif (*Figs.* 479, 480). The rest of the skeleton was radiologically normal.

Blood-picture :—

Hb 72 per cent. Erythrocytes 4,200,000. Leucocytes 7200.

Polymorphonuclears	56 *per cent*
Lymphocytes	30
Eosinophils	10
Hyaline	4

W.R.: Negative before and after provocation.

C.S.F.: Free of any abnormality.

AT OPERATION.—Exploration of the right frontal bone shadow was carried out, and it was noticed that the tissues over it were very vascular; the periosteum and outer table of the frontal bone were found intact; when the outer table was cut through, the diploic spaces were found markedly dilated and full of very vascular tissue, which bled freely. A small piece was removed and submitted to microscopical examination.

Professor Sorour Bey reported: The section sent shows normal bony trabeculæ with capillary angiomatosis in the intervening fibrous tissue. *Fig.* 481 is a microphotograph from this case demonstrating these findings.

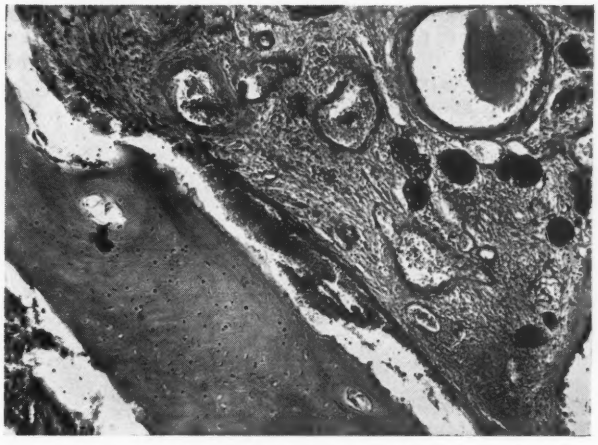


FIG. 481.—Microphotograph showing normal bony trabeculæ with capillary angiomatosis in the intervening fibrous tissue.

On account of the multiplicity of the tumour and the extensive lesion produced, excision has been deferred until a course of deep X-ray therapy has been tried.

REFERENCE

SHERMAN, MARY S. (1944), *Arch. Path.*, **38**, 158.

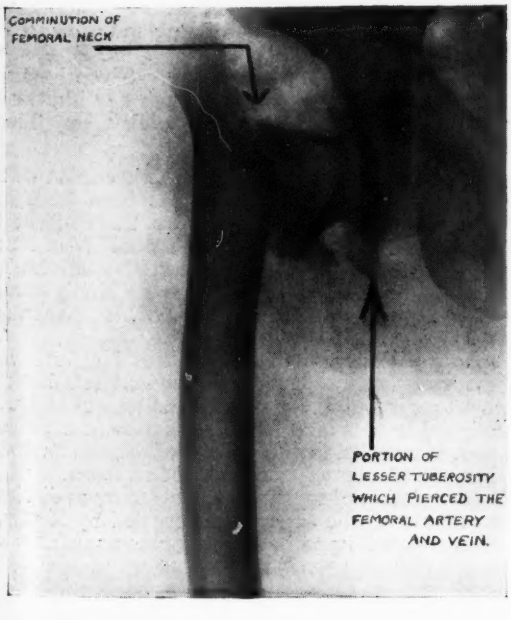
ARTERIOVENOUS ANEURYSM AS A COMPLICATION OF INTERTROCHANTERIC FRACTURE OF THE FEMUR

BY T. A. QUILLIAM

RESIDENT ASSISTANT SURGEON, ST. CHARLES' HOSPITAL, LONDON

CASE REPORT

Mrs. M. H., aged 86 years, was admitted to this hospital on Dec. 19, 1945, having fallen and injured her right hip earlier on the same day.



An intertrochanteric fracture of the right femur was diagnosed and this was confirmed upon X-ray examination, which revealed that considerable comminution of the fragments was present (*Fig.* 482).

A Steinmann pin was inserted through the tibial crest and an extension of 15 lb. was applied with the aid of a Thomas splint and a Pearson bed attachment.

FIG. 482.—Radiograph taken on admission showing comminuted intertrochanteric fracture.

The patient was very senile and was incontinent, but her condition progressed in a fairly satisfactory manner for nearly a month, after which her back and perineum became ulcerated and failed to respond to treatment.

On Jan. 21, 1946, the pin and splint were removed and on Jan. 22, a painless lump made its appearance in the right groin. Arterial pulsation (which could not be obliterated by digital pressure) and a systolic thrill were palpable. Auscultation over the lump in the groin

revealed a continuous 'machinery' murmur. The blood-pressure reading was 120/50 but, unfortunately, no note had been recorded of this previously. The patient died on Jan. 24.

". . . a comminuted fracture was found through the trochanters of the right femur and the lesser trochanter had become almost separated from the rest of the femur, rotated, and driven forward between the femoral artery and vein, penetrating both vessels and forming an arterio-venous aneurysm. The saphenous vein was grossly dilated and packed with blood. There was some extravasation of blood into the tissues of the thigh. . . ."

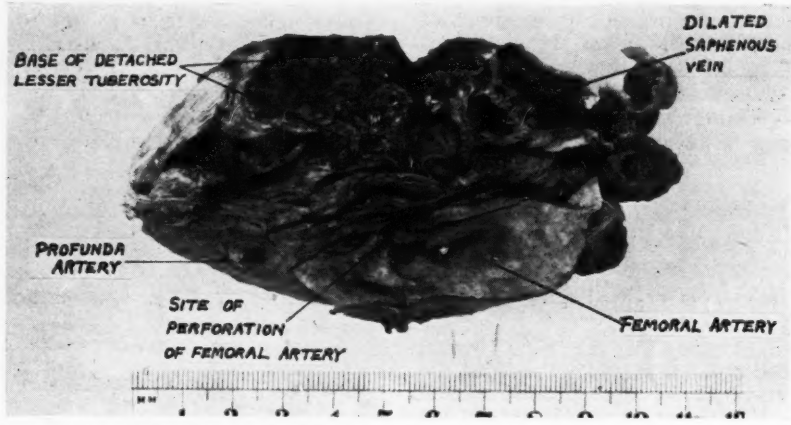


FIG. 483.—Photograph of specimen removed post mortem.

AT AUTOPSY.—An extract of the post-mortem report, by kind permission of Dr. R. D. Teare, is appended, together with a photograph of the specimen removed (*Fig.* 483).

REVIEWS AND NOTICES OF BOOKS

Rœntgen Diagnosis of Diseases of the Gastro-intestinal Tract. By JOHN T. FARRELL, jun., M.D., Clinical Professor of Radiology, Graduate School of Medicine, University of Pennsylvania. 9 × 6 in. Pp. 271 + x, with 190 illustrations. 1946. Springfield, Ill.: C. C. Thomas. (London: Baillière, Tindal & Cox.) 30s.

THIS book contains a large collection of pictures to illustrate all the well-known abnormalities and diseases of the alimentary tract, but it is much more than an atlas of radiography. The text is based upon lecture notes given to post-graduate students in the University of Pennsylvania, and contains valuable and detailed advice about the preparation of patients, the position of the body during examination, and a little about radiological technique. It lays stress upon the methodical observation of certain features such as the contour, mobility, and position of the various organs—a necessary preliminary to the writing of a complete report.

The arrangement of the material follows the pattern of the topographical and aetiological classifications used in the *Standard Classified Nomenclature of Diseases*, and while this has ensured a full consideration of the alimentary canal and made certain that nothing could be omitted, it has resulted in a good deal of overlapping, so that conditions such as cardiospasm and diaphragmatic hernia appear several times over. This is irritating and confusing to the reader, and may even be unreasonable, as when we find duodenal ulcer, because it is classified as a disease due to unknown or uncertain cause, thirty pages after the beginning of the section on the duodenum, with diseases of the small intestine such as ileitis, tuberculosis, and intestinal obstruction interposed.

A book of this type should aim at giving more than a mere description of radiographic outline: for example, it is not enough to state that in the Plummer-Vinson syndrome the thin barium stream may be divided as though cut by a web, without offering any explanation of this phenomenon or any reference as to why the obstruction occurs and how it responds to treatment by iron. Incidentally, it is rather confusing to introduce disorders of the pharynx under the heading 'œsophagus'. No doubt these matters of detail may be rectified in future editions, so that the good promise of the publication may be more completely fulfilled.

Buchanan's Manual of Anatomy. Edited by F. WOOD JONES, D.Sc., F.R.S., F.R.C.S., Sir William Collins Professor of Human and Comparative Anatomy at the Royal College of Surgeons of England; assisted by E. L. PATTERSON, S. MOTTERSHEAD, T. E. BARLOW, F. R. WILDE, and JESSIE DODSON. Seventh edition. 8¾ × 5⅞ in. Pp. 1616 + viii, with 847 illustrations and 48 plates. 1946. London: Baillière, Tindall & Cox. 45s. net.

IT is truly refreshing to see the seventh edition of this famous anatomy text-book, which has been largely rewritten by Wood Jones, assisted by the staff of the Department of Anatomy, University of Manchester. It is, in fact, a new book with new illustrations, new figures, and a new presentation. It was a kindly thought to include a picture of Professor A. M. Buchanan as a frontispiece.

Buchanan's Manual of Anatomy has for forty years been distinguished as being the standard text-book on the gross topographical anatomy of the human body. It was designed to be a guide to the structure of the human body as it is revealed in the process of dissection, and as such has served many generations of students.

The correlation of topographical anatomy as displayed by dissection with the appearance revealed by X rays, is one of the most important developments of modern anatomical teaching, and it is pleasing to see nearly fifty excellent X-ray plates in this new edition.

The glossary has always been a feature of this book and is such a boon to the student, as he is often at a loss to know the meaning of certain anatomical terms. In this edition there are biographical notes of those anatomists whose names occur eponymously in the text; an excellent idea of the new editor.

Students and practitioners the world over are familiar with Wood Jones's other works, especially those on the hand and the foot. In this new edition over 200 drawings by Wood Jones are included in the volume. It is refreshing to see these clear illustrations, which enormously enhance the written work. There are no coloured drawings in the book and we congratulate the editor and publishers in taking this step, for the multicoloured illustrations of anatomy text-books leave much to be desired, for no one ever sees the tissues in these colours, in either the quick or the dead.

We have nothing but praise for this book, and we predict that this seventh edition will maintain the book's firmly established place among English books of anatomy, and we wish it every success.

Gray's Anatomy, Descriptive and Applied. Edited by T. B. JOHNSTON, C.B.E., M.D., Professor of Anatomy in Guy's Hospital Medical School, University of London, and J. WHILLIS, M.D., M.S., Reader in Anatomy, Guy's Hospital Medical School, University of London. Twenty-ninth edition. $9\frac{3}{4} \times 6\frac{1}{8}$ in. Pp. 1597 + xix, with 1359 illustrations, of which 642 are coloured and 47 X-ray plates. 1946. London: Longmans, Green & Co. 70s. net.

THE new Gray will make a strong appeal to all surgeons both at home and abroad because the practical or applied aspect of anatomy is set in its proper place.

The new volume contains some 200 new illustrations, mostly from the pen and brush of that well-known artist, A. K. Maxwell. It is a pity, however, that the poor quality of the paper has prevented the blocks from being good and clear reproductions. Some of the illustrations have merged through the paper and have appeared on the next page.

There is, perhaps, too much small type which is difficult to read. If it is considered by the authors as of little import it would be better omitted.

However, these criticisms are small when the book is considered as a whole, for many generations of surgeons have used it as a reference book and many more will use it in the days to come. To-day it is the anatomical reference book not only of general surgeons but of all surgeons who specialize in the different branches of surgery.

The Treatment of Acute Intestinal Obstruction. By JUDSON T. CHESTERMAN, M.R.C.P. (Lond.), F.R.C.S. (Eng.), F.A.C.S., Surgeon, City General Hospital, Sheffield; Hon. Lecturer, Surgical Pathology, Sheffield University. $8\frac{1}{2} \times 5\frac{3}{8}$ in. Pp. 116 + viii, with 13 illustrations. 1945. London: J. & A. Churchill Ltd. 10s. 6d. net.

THIS small book gives in the compass of a hundred pages a clear account of intestinal obstruction from the viewpoint of interference with intestinal function. The author assumes familiarity with the commoner pathological conditions which cause intestinal obstruction and, although he devotes one-half of his space to the consideration of special obstructions and their treatment, in the main his discussion begins where the text-book stops. His account of the subject is founded on personal clinical experience and experimental observation and has the authority which such a basis gives.

It begins with a consideration of the pathology induced by acute intestinal obstruction and of the additional effects which may follow its release. The results of distension and strangulation were watched through a transparent window in the abdominal wall of cats and these are compared with clinical observations in man.

In the section dealing with diagnosis there is a short, clear description of the value of X-ray films illustrated by plates.

The value of suction in its relation to operation is fully discussed and the operations applicable to different types of obstruction are briefly described.

A short list of references to the more important recent literature is given at the end of each chapter.

Synopsis of Genitourinary Diseases. By AUSTIN I. DODSON, M.D., F.A.C.S., Richmond, Va. Fourth edition. $7\frac{5}{8} \times 4\frac{7}{8}$ in. Pp. 313, with 112 illustrations. 1945. London: Henry Kimpton. 18s. net.

WITHIN the limits imposed by its size, this book contains a vast amount of information; although it is of necessity somewhat condensed, it is still readable. Amongst the chapters to be singled out for particular praise are those on the aetiology and prevention of urinary calculi and on the non-tuberculous infections of the urinary tract; this latter chapter contains useful tables for an acid-ash diet.

We cannot agree with a method of urethral instrumentation which involves a routine change of hands during the manœuvre, but this is probably a relic of the old-fashioned habit of working from the left of the patient.

We can recommend this small book with every confidence for the student who wishes to know the essentials of the subject; it makes a good companion for the author's larger text-book of urological surgery.

Chemotherapy. Yesterday, Today, and Tomorrow. The Linacre Lecture. 1946. By Sir ALEXANDER FLEMING. $7\frac{3}{8} \times 5$ in. Pp. 39, with 5 illustrations and 2 plates. 1946. London: Cambridge University Press. 2s. net.

THIS pamphlet contains an account of Sir Alexander Fleming's experiments to show that the older antiseptics were more toxic to leucocytes than to bacteria; a review of the sulphonamides which, though much more toxic to bacteria than to leucocytes, may have poisonous effects on the whole organism; and the grounds for regarding the newer antibiotics as superior to any other chemotherapeutic agents.

In looking forward to further developments, for "it is the glory of a good bit of work that it opens the way for something still better", an earnest plea is made for the establishment of an institute of microbiology. The answer is to be found in bacterial metabolism. When a bacteriologist discovers an antibiotic in a mould, the chemist should try to isolate the active substance, and if necessary, alter its structure to extract the toxic elements. Team work of this order can flourish only in an institute such as Sir Alexander has in mind.

Technique in Trauma. Planned Timing in the Treatment of Wounds including Burns. From the Montreal General Hospital and McGill University, by FRASER B. GURD, M.D., C.M., and F. DOUGLAS ACKMAN, M.D., C.M., and collaborators. 10 × 7 in. Pp. 68 + vii, with 17 illustrations and 3 coloured plates. 1945. London: William Heinemann (Medical Books) Ltd. 15s. net.

Technique in Trauma is a monograph on the treatment of burns and war wounds emanating from the Montreal Hospital and the University of McGill. Canadian surgery always holds our respect, and the authors, Fraser B. Gurd and F. Douglas Ackman, as well as the publishers, Wm. Heinemann, are to be congratulated on a carefully prepared and well-documented production. Published in 1945, it records the results of treatment by

sulphathiazole emulsion, occlusive compression dressings, and as far as possible unimmobilization. There is a Preface by John Lockwood, and a Commentary by Ralph Fitzgerald, with sections devoted to major and minor burns, the management of burns, and planned timing in the treatment of wounds and infections by the same technique. This book forms a valuable contribution among the many organized schools of thought which originated during the war and were devoted to this subject before the use of penicillin came into general operation. It would seem that the time is ripe for a comprehensive survey of the subject, to record not only the progress that has been made, but to sift the several methods, and pronounce which on the evidence is the most satisfactory for general use.

Practical Points in Penicillin Treatment. By G. E. BEAUMONT, D.M. (Oxon.), F.R.C.P. (Lond.), Physician to the Middlesex Hospital, and K. N. V. PALMER, M.B. (Cantab.), M.R.C.P. (Lond.), Acting Medical Registrar to the Middlesex Hospital. $7\frac{1}{4} \times 4\frac{7}{8}$ in. Pp. 16 + iv, with 1 illustration. 1946. London: J. & A. Churchill. 1s. 6d. net.

THIS booklet sets out in a readily accessible form all the information which House Officers and Practitioners need if they are to use penicillin safely and effectively. A brief but comprehensive account is given of the various preparations, how they are dispensed and stored, and exactly how they are administered. Among many valuable hints the clear directions for giving intramuscular injections call for special attention. The clinical applications of penicillin therapy completes a pamphlet the importance of which can scarcely be over-emphasized now that penicillin is easily obtainable by practitioners who have had no opportunity for special training in its use.

A Complete Outline of Fractures, including Fractures of the Skull. By J. GRANT BONNIN, M.B., B.S. (Melbourne), F.R.C.S. (Eng.). Second edition. $8\frac{5}{8} \times 5\frac{5}{8}$ in. Pp. 658 + xiv, with 712 illustrations. 1946. London: William Heinemann (Medical Books) Ltd. 30s. net.

THIS second edition, though still properly entitled an *Outline of Fractures*, is considerably more bulky than the first; from the student's point of view there is virtue in a book which is reasonably small, yet all the additions to this work are to be welcomed as bare necessities if it is to be kept up to date and remain a useful guide to treatment. The importance of early open reduction and fixation when the circumstances demand it is given fresh emphasis, and the possibilities of operative fixation by modern methods are given due consideration.

The influence of war surgery is evident in this edition, but while much of the advice given about the treatment of lacerated wounds is good, the criticism of the local use of antiseptics is overdone, and the objects of wound excision are not quite correctly expounded. It is courageous to include fractures of the skull, and the author is well qualified to do so because of his special experience in this field. On the other hand, the neurological part of this chapter is too long, requires rearrangement to make it more understandable by the student, and ought to be brought more into harmony with the now generally accepted views about cerebral concussion.

Mr. J. B. Barron's contribution on fractures of the face and jaw is admirable, and the appendices, which include disability tables, surgical exposure of the long bones, and a scheme for the organization of a fracture clinic, present valuable information in a form which is easily available to the student who may have difficulty in consulting the original sources.

The book can be warmly recommended for the help it affords both to those working for examinations and to practitioners. But in addition it records the experience and opinions of a thoughtful and progressive surgeon who can profit from the criticism of his colleagues. The references to the literature of fracture treatment are well chosen, and are therefore of real assistance to any who seek more detailed information.

The Renaissance and Its Influence on English Medicine, Surgery, and Public Health. By Sir ARTHUR SALUSBURY MACNALTY, K.C.B., M.D., F.R.C.P., F.R.C.S. Being the Thomas Vicary Lecture. $8\frac{1}{8} \times 5\frac{3}{8}$ in. 32 pp., with 4 reproductions. 1946. London: Christopher Johnson. 5s. net.

THIS small monograph contains the Thomas Vicary Lecture which was delivered at the Royal College of Surgeons of England in November, 1945.

The author gives an excellent account of medical men in the sixteenth century and their royal patients. Thomas Vicary lived in an interesting age in which Linacre, Erasmus, Dean Colet, Sir Thomas More, Sir Thomas Elyot, and Andrew Boorde held the stage.

A Manual of Tomography. By M. WEINBREN, B.Sc., M.R.C.S., L.R.C.P., F.F.R., D.M.R.E., Lt.-Col. S.A.M.C., Adviser in Radiology, Union Defence Force; Radiologist, Chamber of Mines Hospital, Johannesburg. $9\frac{3}{4} \times 7\frac{3}{8}$ in. Pp. 270 + viii, with 397 illustrations in 138 figures. 1946. London: H. K. Lewis & Co. Ltd. 45s. net.

THE author has written this book primarily for those who have no knowledge or experience of tomography. The technique is described and examples given of many cases in which tomography was of value in supplementing the routine radiological examination. The value of tomography in conditions of the lung is well known in this country, but other applications of the method, particularly in the radiological examination of the spine and other bones, are probably not as familiar here as they should be. The author describes many cases in which it was possible to demonstrate fractures or sequestra in the spine and other bones, which were not seen on routine radiographs. He does not give a critical survey of the value of the method to the surgeon, but contents himself with describing a number of cases in which tomography brought to light features invisible on routine radiographs.

The Principles of Anatomy. An Introduction to Human Biology. By A. A. ABBIE, M.D., B.S., D.Sc. (Syd.), Ph.D. (Lond.), Elder Professor of Anatomy and Histology in the University of Adelaide. Second edition, revised and enlarged. $8\frac{1}{2} \times 5\frac{3}{8}$ in. Pp. 273 + x, with 76 illustrations. 1946. Sydney and London: Angus & Robertson Ltd. 12s. 6d. net.

THIS is a very remarkable book. It was originally written to show how biology could be taught as well from the human as from any other animal, but it has such solid foundations, the details have been so clearly filled in, and the illustrations are so informative that this second edition now contains all that the well-trained medical man knows of human anatomy. Yet it is in no sense of the term a 'cram' book, and the author's intention is that it shall act as an introduction to rather than as a text-book of human anatomy.

By wise choice and skilful presentation of his subjects Professor Abbie has shown how the essentials of anatomy can be offered to the student in a form which is less bulky and more easily assimilated than the standard text-books. Since it seems to be necessary to teach a lot if a little is to be learnt, the big books will no doubt remain with us indefinitely; but it is to be hoped that this book may give further support to the present tendency to relieve the student of an unnecessarily heavy burden in his pre-clinical years.

The Principles of Neurological Surgery. By LOYAL DAVIS, M.S., M.D., Ph.D., D.Sc. (Hon.), Professor of Surgery and Chairman of the Division of Surgery, Northwestern Medical School, Chicago, Illinois. Third edition, thoroughly revised. 9⅛ × 5⅞ in. Pp. 540, with 348 illustrations and 5 plates. 1946. London: Henry Kimpton. 37s. 6d. net.

It is only four years since the second edition of this monograph appeared, yet in that intervening period a large amount of new work has appeared in the realm of neurological surgery. There has been much new and experimental work on the mechanics of craniocerebral injuries and the practical lessons which have accrued from the second world war are far-reaching in their importance.

The new third edition has been very thoroughly revised and brought up to date and can be recommended as a most useful book for students and post-graduates who wish to study neurological surgery.

The Surgical Clinics of North America. Nation-wide Number, October, 1946. Symposia on Aseptic Surgical Technique and its Application to Thoracic Surgery, Gastrointestinal Surgery, and Genitourinary Surgery. 9 × 6 in. Pp. 269 + vi, with 93 illustrations. London and Philadelphia: W. B. Saunders Co. Annual subscription (six numbers), paper 55s.; cloth 75s.

THIS volume contains symposia on antiseptic surgical technique, gastro-intestinal surgery, and genito-urinary surgery. Each symposium contains about six articles covering each subject.

The article on the historical aspects of aseptic surgical technique is well written and illustrated. The value of ultra-violet irradiation of the air of the operating theatre is stressed. The importance of pre-operative skin preparation is discussed at length as it is considered to be the weakest link in the chain of sterile surgical technique.

Other articles are concerned with chest injuries, plastic surgery of the breast, penetrating wounds of the abdomen, anastomosis of the colon, tests for renal function, and the pathology of genito-urinary neoplasms.

This volume contains some stimulating and interesting articles which should prove of real value to surgeons.

Further Studies in Encephalography. By E. GRAEME ROBERTSON, M.D. (Melb.), F.R.C.P., F.R.A.C.P., Neurologist, Royal Melbourne Hospital; etc. 9¾ × 7¼ in. Pp. 104 + x, with 54 illustrations, many as plates. 1946. Melbourne: Macmillan & Co. Ltd. 35s.

IT was only in 1941 that the author of this book published his well-known monograph on Encephalography, and since that date he has not only perfected his technique but also his diagnostic powers.

The present volume, which is dedicated to Dr. Gordon Holmes, contains a wealth of information on experimental studies and clinical studies in ventricular filling.

The factors which may prevent ventricular filling are well discussed and the importance of correct posture is stressed.

The radiographs, which are numerous, are of a high order and greatly enhance the written word.

There can be no doubt whatever that if encephalography is improved still further it will entirely eliminate ventriculography.

This book will interest the neurologist, neurosurgeon, and the radiologist, for it contains new facts and new ideas.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

Human Torulosis. A Clinical, Pathological, and Microscopical Study, with a Report of Thirteen Cases. By LEONARD B. COX, M.D. (Melb.), M.R.C.P. (Edin.), F.R.A.C.P., Hon. Neurologist, the Alfred Hospital, Melbourne; Lecturer in Neuropathology, University of Melbourne; and JEAN C. TOLHURST, M.Sc. (Melb.), Senior Bacteriologist to the Alfred Hospital, Melbourne. 9¾ × 7¼ in. Pp. 149 + xi, with 67 illustrations and 15 plates. 1946. Melbourne: Melbourne University Press. (London: Oxford University Press.) 25s. net.

Physics for the Anæsthetist. By R. R. MACINTOSH, M.A., M.D., F.R.C.S., D.A., Nuffield Professor of Anæsthetics, University of Oxford; etc.; and WILLIAM W. MUSHIN, M.A., M.B., B.S., D.A., First Assistant, Nuffield Department of Anæsthetics, University of Oxford; etc. 8½ × 5½ in. Pp. 235 + viii, with 282 illustrations by Miss M. MCLARTY 1946. Oxford: Blackwell Scientific Publications Ltd. 30s. net.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE MCGREGOR, M.Ch. (Edin.), F.R.C.S. (Eng.), Surgeon, Johannesburg General Hospital; Lecturer on Surgical Anatomy, University of Witwatersrand. With a Foreword by Sir HAROLD J. STILES, K.B.E., F.R.C.S. (Edin.). Sixth edition. 7¼ × 4¾ in. Pp. 714 + xvi, with 699 illustrations. Bristol: John Wright & Sons Ltd. 25s. net.

A Descriptive Atlas of Radiographs. An Aid to Modern Clinical Methods. By A. P. BERTWISTLE, M.B., Ch.B., F.R.C.S. (Edin.). Sixth edition. 9¾ × 7⅛ in. Pp. 606 + xxix, with 948 illustrations. 1946. London: Henry Kimpton. 45s. net.

A Short Handbook of Practical Anæsthetics. By HOEL PARRY-PRICE, M.R.C.S., L.R.C.P., D.A. (R.C.S.), Late Senior Anæsthetist, Royal Berkshire Hospital; Anæsthetic Specialist to the Royal Navy; Surgeon Commander, V.D., R.N.V.R. With a Foreword by Sir CECIL WAKELEY, Consulting Surgeon to the Royal Navy. 7¼ × 4⅞ in. Pp. 127 + viii, with 50 illustrations. 1946. Bristol: John Wright & Sons Ltd. 12s. 6d. net.

Urologic Roentgenology. By MILEY B. WESSON, M.D., Ex-President, American Urological Association. Second edition, thoroughly revised. 9⅛ × 5⅞ in. Pp. 259, with 258 illustrations. 1946. London: Henry Kimpton. 27s. 6d. net.

The Centennial of Surgical Anesthesia. An Annotated Catalogue of Books and Pamphlets bearing on the Early History of Surgical Anesthesia. Exhibited at the Yale Medical Library, October, 1946. Compiled by JOHN F. FULTON, M.D., and MADELINE E. STANTON, A.B. 9 × 6 in. Pp. 102 + xv, with Frontispiece. 1946. New York: Henry Schuman. $4.00.

A Memoir to the Academy of Sciences at Paris on a New Use of Sulphuric Ether. By W. T. G. MORTON, of Boston, in the U.S.A. Presented by M. ARAGO in the Autumn of 1847. With a Foreword by JOHN F. FULTON. 9 × 6 in. Pp. 24 + vi. 1946. New York: Henry Schuman. $1.50.

The Pathology of Traumatic Injury. A General Review. By JAMES V. WILSON, M.D., M.R.C.P. (Lond.), Major, R.A.M.C. (T.). Pathologist to Harrogate and District Hospital and the Royal Bath Hospital, Harrogate. With a Foreword by PHILIP H. MITCHINER, C.B., C.B.E., T.D., M.D., M.S., F.R.C.S. D.Ch., K.H.S. 9⅝ × 6¾ in. Pp. 192 + xii, with 61 illustrations. 1946. Edinburgh: E. & S. Livingstone Ltd. 20s. net.

Urgent Surgery. Edited by JULIUS L. SPIVACK, M.D., LL.D., Associate Professor of Surgery, University of Illinois College of Medicine; etc. 9¾ × 6¾ in. Volume I. Pp. 714 + ix, with 244 illustrations, 14 in colour. 1946. Springfield, Ill.: Charles C. Thomas. $10.00.

Early Ambulation and Related Procedures in Surgical Management. By DANIEL J. LEITHAUSER, M.D., F.A.C.S., Chief of Surgery, St. Joseph Mercy Hospital, Detroit, Michigan. 9 × 6 in. Pp. 232 + xi, with 36 illustrations. 1946. Springfield, Ill.: Charles C. Thomas.

L'Ostéosynthèse au Clou. By R. SŒUR, Chirurgien-Adjoint des Hôpitaux de Bruxelles. 9½ × 6¾ in. Pp. 132, with 134 illustrations. 1946. Paris: Masson et Cie. 325 fr.

Minor Surgery (Heath: Pollard: Davis: Williams). For the Use of House Surgeons, Dressers, and Junior Practitioners. By CECIL FLEMMING, O.B.E., M.Ch., F.R.C.S., Surgeon, University College Hospital. With a chapter on The Administration of Anæsthetics by H. N. WEBBER, B.Ch., D.A., Anæsthetist, University College Hospital. Twenty-third edition. 7¼ × 4¾ in. Pp. 406 + viii, with 209 illustrations. 1946. London: J. & A. Churchill Ltd. 14s. net.

Ambulatory Proctology. By ALFRED J. CANTOR, M.D., Associate Proctologist, Kew Gardens General Hospital, Long Island, New York. With a Foreword by BEAUMONT S. CORNELL, M.D., Editor, *American Journal of Digestive Diseases*. 9 × 6⅛ in. Pp. 524 + xviii, with 281 illustrations. 1946. London: Hamish Hamilton Ltd. 42s. net.

Renal Diseases. By E. T. BELL, M.D., Professor of Pathology in the University of Minnesota, Minneapolis Minnesota. 9⅛ × 5¾ in. Pp. 434, with 115 illustrations and 4 coloured plates. 1946. London: Henry Kimpton. 35s. net.

The Chemistry of Anæsthesia. By JOHN ADRIANI, M.D., Director, Department of Anæsthesia, Charity Hospital of Louisiana at New Orleans; etc. 8¾ × 5½ in. Pp. 530 + ix, with 45 illustrations. 1946. Oxford: Blackwell Scientific Publications. 35s. net.

Confrontations radio-anatomo-cliniques. Publiées sous la direction de M. CHIRAY, R.-A.GUTMANN, et T. Sénèque. Fasicule I. 12½ × 9¾ in. Pp. 56, with 98 illustrations. 1946. Paris: G. Doin et Cie, Masson et Cie. 370 fr.